EVANS ROLLINS CS

```
Dialog level 98.01.01D

Last logoff:  27jan98 13:42:54
Logon file001 28jan98 09:53:37
ANNOUNCEMENT    ****     ANNOUNCEMENT    ****     ANNOUNCEMENT
NEW
***TableBase (File 93)
***U.S. Newswire (File 605)
***OneSearch REPORT TITLES available in Market Research Files
***DIALOG Direct(SM) Launched!
                              ***

RELOADS
***BioCommerce Abstracts and Directory, File 286
***IMSWorld Patents International, Files 447 and 947
***CLAIMS/U.S. PATENTS (File 340): The complete patent collection
   is now in a single file (Dialog File 340) which incorporates
   the following discontinued CLAIMS files: 125,23,24,25. Updates
   are now weekly.
***CLAIMS/UNITERM (File 341) now incorporates the following
   discontinued CLAIMS files: 223,224,225.
***CLAIMS/COMPREHESIVE (File 942) now incorporates the following
   discontinued files: 923,924,925.
                              ***

FORMAT CHANGES
***Derwent World Patents Index (Files 351/352) display
   formats have changed. See HELP NEWS351.
                              ***

DIALOG ONDISC(TM)
***New Dialog OnDisc(TM):  British Education Index
                              ***

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   January 31 and pay only $199. April 15-17 in Philadelphia.
                              ***

PRICE CHANGES
***Prices have been adjusted in a number of Dialog databases
   as of January 1. Updated price list is available via
   ASAF (document numbers 5008-5011) and on the Web at
   http://phoenix.dialog.com/products/dialog/dial_pricing.html.

     >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
     >>>    of new databases, price changes, etc.      <<<
     >>>    Announcements last updated   27Jan98       <<<
* * * New CURRENT year ranges installed.* * *


File   1:ERIC  1966-1997/Nov
       (c) format only 1998 The Dialog Corporation

      Set  Items  Description
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            $0.06    0.002 Hrs File1
     $0.06  Estimated cost File1
     $0.06  Estimated cost this search
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     $0.06  Estimated total session cost   0.002 Hrs.

File 410:Chronolog(R)  1981-1997/Dec
       (c) 1998 Dialog Corporation

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HILIGHT set on as ''
? begin 55,72,154,399,351

       28jan98 09:53:55 User208760 Session D955.2
            $0.00    0.002 Hrs File410
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     $0.00  Estimated cost this search
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SYSTEM:OS  - DIALOG OneSearch
  File  55:BIOSIS PREVIEWS(R)  1985-1998/Jan W4
        (c) 1998 BIOSIS
  File  72:EMBASE  1985-1997/Dec W2
        (c) 1998 Elsevier Science B.V.
*File  72: EMTAGS no longer in EMBASE as of 1/98.  Type: HELP NEWS 72
                        for details.
  File 154:MEDLINE(R)  1985-1998/Mar W2
        (c) format only 1998 Dialog Corporation
  File 399:CA SEARCH(R)  1967-1998/UD=12804
        (c) 1998 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
RANK charge added; see HELP RATES 399.
  File 351:DERWENT WPI  1963-1997/UD=9803;UP=9751;UM=9749
        (c)1998 Derwent Info Ltd
*File 351: Enter HELP NEWS 351 for info. about changes in DWPI coverage.
Output formats have changed for 1998.  Enter HELP FORM351 for details.

      Set  Items  Description
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? s 561(w)1 and antibod?

Processing
Processing
          2353  561
       8971149  1
            17  561(W)1
       1008678  ANTIBOD?
      S1       0  561(W)1 AND ANTIBOD?
? s 5G1(w)1 and antibod?

            21  5G1
       8971149  1
             5  5G1(W)1
       1008678  ANTIBOD?
      S2       5  5G1(W)1 AND ANTIBOD?
? rd s2

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S3       2  RD S2 (unique items)
? t s3/7/all
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3/7/1 (Item 1 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

13595493 BIOSIS Number: 99595493
Inhibition of complement activity by humanized anti-C5 **antibody** and single-chain Fv
Thomas T C; Rollins S A; Rother R P; Giannoni M A; Hartman S L; Elliott E A; Nye S H; Matis L A; Squinto S P; Evans M J
Alexion Pharmaceuticals, 25 Science Park, New Haven, CT 06511, USA
Molecular Immunology 33 (17-18). 1996 (1997). 1389-1401.
Full Journal Title: Molecular Immunology
ISSN: 0161-5890
Language: ENGLISH
Print Number: Biological Abstracts Vol. 104 Iss. 002 Ref. 021507
Activation of the complement system contributes significantly to the pathogenesis of numerous acute and chronic diseases. Recently, a monoclonal **antibody** (**5G1.1**) that recognizes the human complement protein C5, has been shown to effectively block C5 cleavage, thereby preventing the generation of the pro-inflammatory complement components C5a and C5b-9. Humanized **5G1.1 antibody** , Fab and scFv molecules have been produced by grafting the complementarity determining regions of **5G1.1** on to human framework regions. Competitive ELISA analysis indicated that no framework changes were required in the humanized variable regions for retention of high affinity binding to C5, even at framework positions predicted by computer modeling to influence CDR canonical structure. The humanized Fab and scFv molecules blocked complement-mediated lysis of chicken erythrocytes and porcine aortic endothelial cells in a dose-dependent fashion, with complete complement inhibition occurring at a three-fold molar excess, relative to the human C5 concentration. In contrast to a previously characterized anti-C5 scFv molecule, the humanized h5G1.1 scFv also effectively blocked C5a generation. Finally, an intact humanized h5G1.1 **antibody** blocked human complement lytic activity at concentrations identical to the original murine monoclonal **antibody**. These results demonstrate that humanized h5G1.1 and its recombinant derivatives retain both the affinity and blocking functions of the murine **5G1.1 antibody**, and suggest that these molecules may serve as potent inhibitors of complement-mediated pathology in human inflammatory diseases.

3/7/2 (Item 1 from file: 351)
DIALOG(R)File 351:DERWENT WPI

010491522
WPI Acc No: 95-392923/199550
Treating glomerulonephritis with **antibody** against complement C5 component - to inhibit complement induced cell lysis
Patent Assignee: ALEXION PHARM INC (ALEX-N)
Inventor: EVANS M J; MATIS L; MUELLER E E; NYE S H; ROLLINS S; ROTHER R P; SPRINGHORN J P; SQUINTO S P; THOMAS T C; WANG Y; WILKINS J A
Number of Countries: 065 Number of Patents: 004
Patent Family:

Patent No	Kind	Date	Applicat No	Kind	Date	Main IPC	Week
WO 9529697	A1	19951109	WO 95US5688	A	19950501	A61K-038/36	199550 B
AU 9524747	A	19951129	AU 9524747	A	19950501	A61K-038/36	199609
EP 758904	A1	19970226	EP 95919041	A	19950501	A61K-038/36	199714
			WO 95US5688	A	19950501		
BR 9507594	A	19970916	BR 957594	A	19950501	A61K-038/36	199744
			WO 95US5688	A	19950501		

Priority Applications (No Type Date): US 94236208 A 19940502
Cited Patents: 03Jnl.Ref; US 5135916
Patent Details:

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 Patent   Kind Lan Pg Filing Notes     Application  Patent
 WO 9529697  A1 E 181
    Designated States (National): AM AU BB BG BR BY CA CN CZ EE FI GE HU IS
    JP KG KP KR KZ LK LR LT LV MD MG MN MX NO NZ PL RO RU SG SI SK TJ TM TT
    UA UG US UZ VN
    Designated States (Regional): AT BE CH DE DK ES FR GB GR IE IT KE LU MC
    MW NL OA PT SD SE SZ UG
 AU 9524747  A        Based on                      WO 9529697
 EP 758904   A1 E     Based on                      WO 9529697
    Designated States (Regional): AT BE CH DE DK ES FR GB IE IT LI NL PT SE
 BR 9507594  A        Based on                      WO 9529697

 Abstract (Basic): WO 9529697 A
        Glomerulonephritis (GN) is treated by admin. of an antibody
     (Ab) that binds to complement component C5 in the blood to reduce the
     cell-lysing activity of complement. Also new are: (1) Ab specific for
     the alpha chain of human C5, able to inhibit complement activated lysis
     but unable to bind specifically to the free C5a activation product; (3)
     the hybridoma 5G1.1 (ATCC HB.11625); (4) Abs produced by
     this hybridoma or antibodies able to compete with it for binding
     to C5 alpha chain; (5) a nucleic acid (I) encoding a single chain (sc)
     Fv polypeptide of 248 amino acids.
         USE - The Abs practically eliminate glomerular inflammation and
     enlargement associated with GN, and can also be used wherever
     inhibition of complement is required, e.g. in cases of inflammatory
     joint disease or in treatment of immunological or haematological
     disorders associated with extracorporeal circulation. The isolated
     alpha chain of C5 and peptides can be used to induce prodn. of Ab by
     immunisation, or to screen candidate antibodies for anti-C5
     activity.
         ADVANTAGE - Ab are specific for C5 and do not affect opsonic,
     anti-infective and immune complex clearance functions of complement.
     Some Abs block haemolysis by complement at close to the theoretical 1:2
     antibody:antigen ratio.
         Dwg.0/19
 Derwent Class: B04; D16
 International Patent Class (Main): A61K-038/36
 International Patent Class (Additional): A61K-039/00; A61K-039/395;
   C07K-014/00; C07K-014/75; C07K-016/00; C07K-016/18; C07K-016/36;
   C07K-016/46; C12N-005/10; C12N-005/20; C12N-015/09; C12N-015/10;
   C12N-015/13; C12N-015/63; C12P-021/02; C12P-021/08
 ? s ksskc and (antibod? or complement)

               1  KSSKC
         1008678  ANTIBOD?
          121257  COMPLEMENT
      S4       1  KSSKC AND (ANTIBOD? OR COMPLEMENT)
 ? t s4/3/all


  4/3/1     (Item 1 from file: 399)
 DIALOG(R)File 399:CA SEARCH(R)
 (c) 1998 American Chemical Society. All rts. reserv.

   124127101    CA: 124(10)127101t    PATENT
   Anti-complement C5 antibodies for the treatment of glomerulonephritis and
 other inflammatory diseases
   INVENTOR(AUTHOR): Evans, Mark J.; Matis, Louis; Mueller, Eileen Elliott;
 Nye, Steven H.; Rollins, Scott; Rother, Russell P.; Springhorn, Jeremy P.;
 Squinto, Stephen P.; Thomas, Thomas C.; et al.
   LOCATION: USA
   ASSIGNEE: Alexion Pharmaceuticals, Inc.
   PATENT: PCT International ; WO 9529697 A1  DATE: 951109
   APPLICATION: WO 95US5688 (950501) *US 236208 (940502)
   PAGES: 159 pp.  CODEN: PIXXD2  LANGUAGE: English  CLASS: A61K-038/36A;
```

A61K-039/00B; A61K-039/395B; C07K-014/00B; C07K-014/75B; C07K-016/00B;
C07K-016/18B; C07K-016/36B; C07K-016/46B; C12N-005/10B; C12N-005/20B;
C12N-015/09B; C12N-015/10B; C12N-015/13B; C12N-015/63B; C12P-021/02B;
C12P-021/08B DESIGNATED COUNTRIES: AM; AU; BB; BG; BR; BY; CA; CN; CZ; EE;
FI; GE; HU; IS; JP; KG; KP; KR; KZ; LK; LR; LT; LV; MD; MG; MN; MX; NO; NZ;
PL; RO; RU; SG; SI; SK; TJ; TM; TT; UA; UG; US; UZ; VN
DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB;
GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE;
SN; TD; TG

```
? s c5 and 46(w)(kd or kda)

          19202  C5
         240119  46
          77212  KD
         180258  KDA
           2695  46(W)(KD OR KDA)
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? t s6/3/all


 6/3/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
(c) 1998 BIOSIS. All rts. reserv.

13316913     BIOSIS Number: 99316913
  Expression of glutamyl-tRNA reductase in Escherichia coli
  Chen W; Wright L; Lee S; Cosloy S D; Russell C S
  Dep. Chem., City Coll. N.Y., City Univ. N.Y., Convent Ave. at 138th St.,
New York, NY 10031, USA
  Biochimica et Biophysica Acta 1309 (1-2). 1996.  109-121.
  Full Journal Title:  Biochimica et Biophysica Acta
  ISSN: 0006-3002
  Language: ENGLISH
  Print Number: Biological Abstracts Vol. 103 Iss. 002 Ref. 020027


 6/3/2     (Item 2 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
(c) 1998 BIOSIS. All rts. reserv.

11892810     BIOSIS Number: 98492810
  Isolation and preliminary characterization of cDNA encoding American
cockroach allergens
  Wu C-H; Lee M-F; Liao S-C
  Dep. Med. Research, Taichung Veterans General Hosp., Taichung 407, Taiwan
  Journal of Allergy and Clinical Immunology 96 (3). 1995.  352-359.
  Full Journal Title:  Journal of Allergy and Clinical Immunology
  ISSN: 0091-6749
  Language: ENGLISH
  Print Number: Biological Abstracts Vol. 100 Iss. 010 Ref. 143667


 6/3/3     (Item 1 from file: 72)
DIALOG(R)File  72:EMBASE
(c) 1998 Elsevier Science B.V. All rts. reserv.

8430947   EMBASE No: 92107188
  Activation  of the alternative pathway of complement by monoclonal lambda
```

light chains in membranoproliferative glomerulonephritis
Meri S.; Koistinen V.; Miettinen A.; Tornroth T.; Seppala I.J.T.
Department of Bacteriology and Immunology, University of Helsinki, Haartmaninkatu 3, SF-00290 Helsinki Finland
J. EXP. MED. (USA) , 1992, 175/4 (939-950) CODEN: JEMEA ISSN: 0022-1007
LANGUAGES: English SUMMARY LANGUAGES: English
? t s6/7/3

6/7/3 (Item 1 from file: 72)
DIALOG(R)File 72:EMBASE

8430947 EMBASE No: 92107188
Activation of the alternative pathway of complement by monoclonal lambda light chains in membranoproliferative glomerulonephritis
Meri S.; Koistinen V.; Miettinen A.; Tornroth T.; Seppala I.J.T.
Department of Bacteriology and Immunology, University of Helsinki, Haartmaninkatu 3, SF-00290 Helsinki Finland
J. EXP. MED. (USA) , 1992, 175/4 (939-950) CODEN: JEMEA ISSN: 0022-1007
LANGUAGES: English SUMMARY LANGUAGES: English
Immunopathological evidence suggests that activation of the alternative pathway of complement (AP) is involved in membranoproliferative glomerulonephritis (MPGN) and in immunoglobulin A nephropathy. In this report we describe an AP dysfunction-associated factor that was isolated from the serum and urine of a patient with hypocomplementemic MPGN. Extensive glomerular deposits of C3, properdin, and of the terminal complement components were observed in the kidney of the patient. In her serum the AP hemolytic activity was virtually absent. When mixed with fresh normal serum, the patient's serum induced a 96% C3 conversion during a 30-min incubation at +37degreeC. This activity was found to be due to a circulating factor that by immunochemical characterization proved to be a **46-kD** monoclonal immunoglobulin lambda light (L) chain dimer (lambda(L)). Purified lambda(L), but not control lambda or kappa L chains from patients with L chain disease, activated the AP in a dose- and ionic strength-dependent manner. Functionally, lambda(L) was differentiated from C3 nephritic factor (an autoantibody against the AP C3 convertase, C3bBb) by its inability to bind to and stabilize the C3bBb enzyme. Instead, lambda(L) was observed to interact directly with the AP control factor H. Thus, lambda(L) represents a novel type of immunoglobulin-related AP-activating factor with the capacity to initiate alternative complement pathway activation in the fluid phase.
? s c5a and complement and antibod?

```
          6192  C5A
        121257  COMPLEMENT
       1008678  ANTIBOD?
S7        835  C5A AND COMPLEMENT AND ANTIBOD?
```
? s s7 and cleavage

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           835  S7
        159820  CLEAVAGE
S8          56  S7 AND CLEAVAGE
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? rd s8

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
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...completed examining records
S9 27 RD S8 (unique items)
? t s9/7/all

9/7/1 (Item 1 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

13595493 BIOSIS Number: 99595493
Inhibition of **complement** activity by humanized anti-C5 **antibody** and single-chain Fv
Thomas T C; Rollins S A; Rother R P; Giannoni M A; Hartman S L; Elliott E A; Nye S H; Matis L A; Squinto S P; Evans M J
Alexion Pharmaceuticals, 25 Science Park, New Haven, CT 06511, USA
Molecular Immunology 33 (17-18). 1996 (1997). 1389-1401.
Full Journal Title: Molecular Immunology
ISSN: 0161-5890
Language: ENGLISH
Print Number: Biological Abstracts Vol. 104 Iss. 002 Ref. 021507
Activation of the **complement** system contributes significantly to the pathogenesis of numerous acute and chronic diseases. Recently, a monoclonal **antibody** (5G1.1) that recognizes the human **complement** protein C5, has been shown to effectively block C5 **cleavage**, thereby preventing the generation of the pro-inflammatory **complement** components **C5a** and C5b-9. Humanized 5G1.1 **antibody** , Fab and scFv molecules have been produced by grafting the complementarity determining regions of 5G1.1 on to human framework regions. Competitive ELISA analysis indicated that no framework changes were required in the humanized variable regions for retention of high affinity binding to C5, even at framework positions predicted by computer modeling to influence CDR canonical structure. The humanized Fab and scFv molecules blocked **complement**-mediated lysis of chicken erythrocytes and porcine aortic endothelial cells in a dose-dependent fashion, with complete **complement** inhibition occurring at a three-fold molar excess, relative to the human C5 concentration. In contrast to a previously characterized anti-C5 scFv molecule, the humanized h5G1.1 scFv also effectively blocked **C5a** generation. Finally, an intact humanized h5G1.1 **antibody** blocked human **complement** lytic activity at concentrations identical to the original murine monoclonal **antibody**. These results demonstrate that humanized h5G1.1 and its recombinant derivatives retain both the affinity and blocking functions of the murine 5G1.1 **antibody**, and suggest that these molecules may serve as potent inhibitors of **complement** -mediated pathology in human inflammatory diseases.

9/7/2 (Item 2 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

13446885 BIOSIS Number: 99446885
Monoclonal **antibody** to C5 inhibits **C5a** and C5b-9 generation without inhibition of C3 **cleavage** and significantly limits myocardial ischemia and reperfusion induced tissue damage
Vakeva A; Rollins S A; Matis L A; Stahl G L
Brigham Women's Hosp., Boston, MA, USA
Journal of the American College of Cardiology 29 (2 SUPPL. A). 1997. 267A.
Full Journal Title: 46th Annual Scientific Session of the American College of Cardiology, Anaheim, California, USA, March 16-19, 1997. Journal of the American College of Cardiology
ISSN: 0735-1097
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 049 Iss. 004 Ref. 067737

9/7/3 (Item 3 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

13135752 BIOSIS Number: 99135752

Amelioration of lupus-like autoimmune disease in NZB-W F-1 mice after treatment with a blocking monoclonal **antibody** specific for **complement** component C5

Wang Yi; Hu Q; Madri J A; Rollins S A; Chodera A; Matis L A

Immunobiol. Program, Alexion Pharmaceuticals, Inc., New Haven, CT 06511, USA

Proceedings of the National Academy of Sciences of the United States of America 93 (16). 1996. 8563-8568.

Full Journal Title: Proceedings of the National Academy of Sciences of the United States of America

ISSN: 0027-8424

Language: ENGLISH

Print Number: Biological Abstracts Vol. 102 Iss. 006 Ref. 083883

New Zealand black times New Zealand white (NZB/W) F-1 mice spontaneously develop an autoimmune syndrome with notable similarities to human systemic lupus erythematosus. Female NZB/W F-1 mice produce high titers of antinuclear **antibodies** and invariably succumb to severe glomerulonephritis by 12 months of age. Although the development of the immune-complex nephritis is accompanied by abundant local and systemic **complement** activation, the role of proinflammatory **complement** components in disease progression has not been established. In this study we have examined the contribution of activated terminal **complement** proteins to the pathogenesis of the lupus-like autoimmune disease. Female NZB/W F-1 mice were treated with a monoclonal **antibody** (mAb) specific for the C5 component of **complement** that blocks the **cleavage** of C5 and thus prevents the generation of the potent proinflammatory factors **C5a** and C5b-9. Continuous therapy with anti-C5 mAb for 6 months resulted in significant amelioration of the course of glomerulonephritis and in markedly increased survival. These findings demonstrate an important role for the terminal **complement** cascade in the progression of renal disease in NZB//W F-1 mice, and suggest that mAb-mediated C5 inhibition may be a useful approach to the therapy of immune-complex glomerulonephritis in humans.

9/7/4 (Item 4 from file: 55)

DIALOG(R)File 55:BIOSIS PREVIEWS(R)

13040729 BIOSIS Number: 99040729

Proteolytic inactivation of the leukocyte **C5a** receptor by proteinases derived from Porphyromonas gingivalis

Jagels M A; Travis J; Potempa J; Pike R; Hugli T E

IMM-18, Dep. Immunol., The Scripps Res. Inst., La Jolla, CA 92037, USA

Infection and Immunity 64 (6). 1996. 1984-1991.

Full Journal Title: Infection and Immunity

ISSN: 0019-9567

Language: ENGLISH

Print Number: Biological Abstracts Vol. 102 Iss. 002 Ref. 022902

The anaerobic bacterium Porphyromonas gingivalis has been implicated as a primary causative agent in adult periodontitis. Several proteinases are produced by this bacterium, and it is suggested that they contribute to virulence and to local tissue injury resulting from infection by P. gingivalis. Cysteine proteinases with specificities to cleave either Arg-X or Lys-X peptide bonds (i.e., gingipains) have been characterized as predominant enzymes associated with vesicles shed from the surface of this bacterium. It has recently been demonstrated that these proteinases are capable of degrading the blood **complement** component C5, resulting in the generation of biologically active **C5a** . By using an affinity-purified rabbit **antibody** raised against residues 9 to 29 of the **C5a** receptor (C5aR; CD88), we demonstrate that noncysteinyl proteinases associated with vesicles obtained from P. gingivalis cleave the

C5aR on human neutrophils. Proteolytic attack of the C5aR by enzymes from the P. gingivalis vesicles was inhibited by TPCK (tolylsulfonyl phenylalanyl chloromethyl ketone), PMSF (phenylmethylsulfonyl fluoride), and dichloroisocoumarin, suggesting that serine proteinases are primarily responsible for this degradative activity. The purified vesicle proteinase Lys-gingipain but not Arg-gingipain also cleaved the N-terminal region of the C5aR on the human neutrophils. Lys-gingipain activity was essentially resistant to these inhibitors but was inhibited by TLCK (N-alpha-p-tosyl-L-lysine chloromethyl ketone) and iodoacetamide. A synthetic peptide that mimics the N-terminal region of C5aR (residues 9 to 29; PDYGHY DDKDTLDLNTPVDKT) was readily cleaved by chymotrypsin but not by trypsin, despite the presence of two potential trypsin (i.e., lysyl-X) **cleavage** sites. The specific sites of **cleavage** in the C5aR 9-29 peptide were determined by mass spectroscopy for both chymotrypsin and Lys-gingipain digests. This analysis demonstrated that the C5aR peptide is susceptible to **cleavage** at both potential Lys-gingipain sites (i.e., between residues 17 and 18 (K-D) and 28 and 29 (K-T)) and at two chymotrypsin sites (between residues 14 and 15 (Y-D) and 20 and 21 (L-D)), respectively. These studies suggest that P. gingivalis contains at least two enzymes capable of cleaving the C5aR, Lys-gingipain and a second nontryptic serine proteinase that is distinct from either Arg- or Lys-gingipain.

9/7/5 (Item 5 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

12128182 BIOSIS Number: 98728182

Amplification of the inflammatory response: Adhesion molecules associated with platelet-white cell responses

Rinder C; Fitch J

Dep. Anesthesia, Yale University, 333 Cedar Street, PO Box 3333, New Haven, CT 06510, USA

Journal of Cardiovascular Pharmacology 27 (SUPPL. 1). 1996. S6-S12.

Full Journal Title: Journal of Cardiovascular Pharmacology

ISSN: 0160-2446

Language: ENGLISH

Print Number: Biological Abstracts Vol. 101 Iss. 008 Ref. 112457

Cardiopulmonary bypass (CPB) causes leukocyte and platelet activation, resulting in upregulation of the adhesion receptor CD11b/CD 1 8 on leukocytes and upregulation of P-selectin, the adhesion receptor that binds the activated platelet to polymorphonuclear neutrophils (PMNs) and monocytes. Our laboratory has studied the expression of activation-dependent adhesion receptors during in vivo CPB. Both PMN and monocyte CD11b were upregulated during CPB but with differing time courses. Peak PMN CD11b levels occurred at the end of the hypothermic phase of bypass, whereas monocyte CD11b levels increased steadily throughout the course of CPB, peaked at 2-4 h after CPB, and remained significantly elevated as late as 18-24 h post CPB. The percentage of P-selectin-positive platelets increased significantly during bypass, peaking around the end of bypass and remaining elevated in the early post-bypass period. The level then returned to normal by 18 h post-bypass. Monocyte-platelet binding paralleled the increase in P-selectin-positive platelets during bypass and similarly remained elevated in the post-bypass period. PMN-platelet binding also increased but peaked early during CPB. Upregulation of these adhesive receptors and formation of platelet-leukocyte conjugates may influence the prothrombotic activity of monocytes and the proinflammatory activity of PMNs in the post-CPB period. Our laboratory has developed an in vitro model of extracorporeal circulation, and recirculation of blood on this circuit results in significant activation of PMNs and monocyte CD11b expression, increasing progressively over time. Likewise, the percentage of P-selectin-positive platelets increased and was paralleled by the formation of leukocyte-platelet conjugates comparable to the pattern found in vivo. Generation of the **complement** fragments **C5a** and the C5b-9

membrane-attack complex may contribute to platelet P-selectin expression and formation of leukocyte-platelet conjugates during CPB. The in vitro model has been used to test the cellular effects of **complement** inhibition employing a monoclonal **antibody** that blocks **cleavage** of C5 into **C5a** and C5b to determine the role of early vs. late **complement** components in the cellular activation induced by CPB. Preliminary results demonstrate that blockade of the formation of **C5a** and the C5b-9 membrane-attack complex during simulated extracorporeal circulation effectively inhibits platelet and PMN activation and the formation of leukocyte-platelet conjugates.

9/7/6 (Item 6 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

12080123 BIOSIS Number: 98680123
In vitro and in vivo inhibition of **complement** activity by a single-chain Fv fragment recognizing human C5
Evans M J; Rollins S A; Wolff D W; Rother R P; Norin A J; Therrien D M; Grijalva G A; Mueller J P; Nye S H; Squinto S P; Wilkins J A
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Molecular Immunology 32 (16). 1995. 1183-1195.
Full Journal Title: Molecular Immunology
ISSN: 0161-5890
Language: ENGLISH
Print Number: Biological Abstracts Vol. 101 Iss. 006 Ref. 080404
Complement activation has been implicated in the pathogenesis of several human diseases. Recently, a monoclonal **antibody** (N19-8) that recognizes the human **complement** protein C5 has been shown to effectively block the **cleavage** of C5 into **C5a** and C5b, thereby blocking terminal **complement** activation. In this study, a recombinant N19-8 scFv **antibody** fragment was constructed from the N19-8 variable regions, and produced in both mammalian and bacterial cells. The N19-8 scFv bound human C5 and was as potent as the N19-8 monoclonal **antibody** at inhibiting human C5b-9-mediated hemolysis of chicken erythrocytes. In contrast, the N19-8 scFv only partially retained the ability of the N19-8 monoclonal **antibody** to inhibit **C5a** generation. To investigate the ability of the N19-8 scFv to inhibit **complement**-mediated tissue damage, **complement** -dependent myocardial injury was induced in isolated mouse hearts by perfusion with Krebs-Henseleit buffer containing 6% human plasma. The perfused hearts sustained extensive deposition of human C3 and C5b-9, resulting in increased coronary artery perfusion pressure, end-diastolic pressure, and a decrease in heart rate until the hearts ceased beating approximately 10 min after the addition of plasma. Hearts treated with human plasma supplemented with either the N19-8 monoclonal **antibody** or the N19-8 scFv did not show any detectable changes in cardiac performance for at least 1 hr following the addition of plasma. Hearts treated with human plasma alone showed extensive deposition of C3 and C5b-9, while hearts treated with human plasma containing the N19-8 scFv showed extensive deposition of C3, but no detectable deposition of C5b-9. Administration of a 100 mg bolus dose of N19-8 scFv to rhesus monkeys inhibited the serum hemolytic activity by at least 50% for up to 2 hr. Pharmacokinetic analysis of N19-8 scFv serum levels suggested a two-compartment model with a T-1/2-alpha of 27 min. Together, these data suggest the recombinant N19-8 scFv is a potent inhibitor of the terminal **complement** cascade and may have potential in vivo applications where short duration inhibition of terminal **complement** activity is desirable.

9/7/7 (Item 7 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

12033363 BIOSIS Number: 98633363

Complement inhibition with an anti-C5 monoclonal **antibody** prevents acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation

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Transplantation (Baltimore) 60 (11). 1995. 1194-1202.

Full Journal Title: Transplantation (Baltimore)

ISSN: 0041-1337

Language: ENGLISH

Print Number: Biological Abstracts Vol. 101 Iss. 004 Ref. 049108

Prevention of hyperacute xenograft rejection in the pig-to-primate combination has been accomplished by removal of natural **antibodies**, **complement** depletion with cobra venom factor, or prevention of C3 activation with the soluble **complement** inhibitor sCR1. Although these strategies effectively prevent hyperacute rejection, they do not address the relative contribution of early (C3a, C3b) versus late (**C5a**, C5b-9) activated **complement** components to xenogeneic organ damage. To better understand the role of the terminal **complement** components (**C5a** , C5b-9) in hyperacute rejection, an anti-human C5 mAb was developed and tested in an ex vivo model of cardiac xenograft rejection. In vitro studies demonstrated that the anti-C5 mAb effectively blocked C5 **cleavage** in a dose-dependent manner that resulted in complete inhibition of both **C5a** and C5b-9 generation. Addition of anti-C5 mAb to human blood used to perfuse a porcine heart prolonged normal sinus cardiac rhythm from a mean time of 25.2 min in hearts perfused with unmodified blood to 79,296, or gt 360 min when anti-C5 mAb was added to the blood at 50 mu-g/ml, 100 mu-g/ml, or 200 mu-g/ml, respectively. In these experiments, activation of the classical **complement** pathway was completely inhibited. Hearts perfused with blood containing the highest concentration of anti-C5 mAb had no histologic evidence of hyperacute rejection and no deposition of C5b-9. These experiments suggest that the activated terminal **complement** components **C5a** and C5b-9, but not C3a or C3b, play a major role in tissue damage in this porcine-to-human model of hyperacute rejection. They also suggest that targeted inhibition of terminal **complement** activation by anti-C5 mAbs may be useful in clinical xenotransplantation.

9/7/8 (Item 8 from file: 55)

10845849 BIOSIS Number: 97045849

Rapid quantification of C3a and **C5a** using a combination of chromatographic and immunoassay procedures

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Journal of Immunological Methods 166 (1). 1993. 35-44.

Full Journal Title: Journal of Immunological Methods

ISSN: 0022-1759

Language: ENGLISH

Print Number: Biological Abstracts Vol. 097 Iss. 003 Ref. 029735

Monoclonal **antibodies** were isolated which reacted specifically with the **complement** **cleavage** products C3a, C3adR, **C5a**, and C5adR but not with the parent molecules C3 or C5. In both cases the mAbs showed a higher affinity towards the desArg forms. These mAbs were used as capture **antibodies** in immunoassays for C3a/C3adR and **C5a**/C5adR. The immunoassays are based on the ABICAP technology which ensures for a rapid measurement. Due to the large binding capacity and the very short diffusion pathways in the gel-matrix the binding equilibrium between

capture **antibodies** and the antigen is reached whilst the sample is flowing through the column. Therefore this test represents an endpoint assay offering the possibility of using a single calibration curve for a large number of measurements. With the C3adR assay concentrations down to 16 ng/ml C3adR can be detected. The lower detection limit of the C5adR assay is 1 ng/ml C5adR. The tests for C3a/C3adR, and **C5a**/C5adR can be performed in 20 to 25 min and this rapid processing of plasma samples should permit the application of these parameters for diagnostic purposes and patient management.

9/7/9 (Item 9 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

9044010 BIOSIS Number: 93029010
THE ROLE OF **C5A** AND **ANTIBODY** IN THE RELEASE OF HEPARAN SULFATE FROM ENDOTHELIAL CELLS
PLATT J L; DALMASSO A P; LINDMAN B J; IHRCKE N S; BACH F H
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EUR J IMMUNOL 21 (11). 1991. 2887-2890. CODEN: EJIMA
Full Journal Title: European Journal of Immunology
Language: ENGLISH
The activation of endothelial cells is thought to contribute to the host response to infection and to the pathogenesis of autoimmune disease. It was recently shown that **antibody** and **complement** can activate endothelial cells leading to **cleavage** and released of heparan sulfate from the cells. We show here that release of heparan sulfate from endothelial cells is mediated by **antibody** and the **complement** fragment **C5a** and that assembly of the membrane attack complex and lysis of endothelial cells is not necessarily involved. These data suggest that the generation of **C5a** in conditions such as autoimmunity and infection in which anti-endothelial cells **antibodies** may also be present, might amplify tissue injury by a novel mechanism involving endothelial cell activation and loss of heparan sulfate mediated by **antibody** and **C5a**.

9/7/10 (Item 10 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

7364099 BIOSIS Number: 89015118
COMPLEMENT ACTIVATION BY THE ALTERNATIVE PATHWAY IS MODIFIED IN RENAL FAILURE THE ROLE OF FACTOR D
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CLINIQUE MEDICALE, DEP. MED., HOPITAL CANTONAL UNIVERSITAIRE, CH-1211 GENEVA 4, SWITZERLAND.
CLIN NEPHROL 32 (4). 1989. 185-193. CODEN: CLNHB
Full Journal Title: Clinical Nephrology
Language: ENGLISH
Factor D, an essential enzyme of the alternative pathway (AP) of **complement**, is eliminated by the kidney, and its plasma concentration increases 10-fold in end-stage renal disease (ESRD). The purpose of this study was to analyze the consequences of factor D accumulation. A number of in vitro assays were used to analyze AP activation in normal human serum (NHS) in normal serum supplemented with purified factor D to 10-fold its normal concentration (10 .times. D), and in sera of patients with ESRD. When compared with NHS, in 10 .times. D: 1) Spontaneous fluid-phase activation of **complement** at 37.degree.C was greatly increased as measured by C3 **cleavage** , 2) The lysis of rabbit ethrocytes, a function of the AP, was accelerated, 3) More C3 fragments bound to cuprophane membranes and to immune precipitates; both reactions were accompanied by the formation of more **C5a**, 4) **Complement**

mediated solubilization of antigen-**antibody** precipitates was enhanced. Sera of patients with ESRD behaved similarly to 10 .times. D in all assays used, i.e., enhanced AP function, although **complement** activation measured in these assays varied widely from one individual to another. Thus, the elevated factor D concentration observed in renal failure might have important pathophysiological consequences, some of which could be detrimental (e.g., **C5a** produced during hemodialysis), while others might be beneficial, e.g., solubilization of immune precipitates.

9/7/11 (Item 11 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

6956340 BIOSIS Number: 87016861
RELATIVE INEFFICIENCY OF TERMINAL **COMPLEMENT** ACTIVATION
BHAKDI S; FASSBENDER W; HUGO F; CARRENO M-P; BERSTECHER C; MALASIT P; KAZATCHKINE M D
INST. MED. MICROBIOLOGY, UNIV. GIESSEN, SCHUBERTSTRASSE 1, D-6300 GIESSEN, WEST GERMANY.
J IMMUNOL 141 (9). 1988. 3117-3122. CODEN: JOIMA
Full Journal Title: Journal of Immunology
Language: ENGLISH
The efficiency of generation of fluid-phase SC5b-9 and membrane C5b-9(m) complexes relative to **cleavage** of C3 and C5 was studied. Fluid-phase C activation was induced through addition of purified bacterial Ag to human serum. Sephadex beads were used as particulate activators of the alternative pathway. Rabbit or **antibody**-coated sheep or human E were used to study formation of cytolytic C5b-9(m) complexes. The molar ratios of C3a:**C5a** generated in the model systems were found to be in the range of 60 to 200:1 in the case of soluble immune complex activators, and 70 to 150:1 with particulate activators and cells. The efficiency of C5 **cleavage** relative to C3 **cleavage** increased on surfaces with the density of **antibody** and/or C3b-binding sites. With soluble immune complexes, the efficiency of subsequent SC5b-9 generation displayed wide variations dependent on Ag and donor with molar ratios of **C5a**:SC5b-9 ranging from 30:1 for teichoic acid and sometimes approaching 1:1 for streptolysin-O. In contrast, activation on particles or cells always led to **C5a** :C5b-9 (calculated as the sum of generated moles SC5b-9 and C5b-9(m)) ratios approaching 1:1. Hence, there is an overall inefficiency of terminal sequence activation in the C cascade due first to a dissociation at the level of C5 convertase formation/C5-**cleavage** and second, to a frequent inefficiency of C5b-utilization in the fluid-phase. The results provide an explanation for the very low levels of SC5b-9 found in plasma of healthy individuals and in patients with C-consuming immune complex disease.

9/7/12 (Item 12 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

6621576 BIOSIS Number: 86088127
FUNCTIONAL AND BIOCHEMICAL PROPERTIES OF RAT KUPFFER CELLS AND PERITONEAL MACROPHAGES
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J LEUKOCYTE BIOL 44 (2). 1988. 71-78. CODEN: JLBIE
Full Journal Title: Journal of Leukocyte Biology
Language: ENGLISH
Functional and biochemical techniques were used to further characterize heterogeneity between rat Kupffer cells and peritoneal macrophages. Both macrophage cell types were found to phagocytize **antibody** coated sheep red blood cells in a time-dependent manner. However, Kupffer cells were two to three times more phagocytic than were peritoneal macrophages. In

contrast, the peritoneal cells released significantly more superoxide anion in response to the **complement cleavage** product, **C5a** and the phorbol ester tumor promoter, 12-O-tetradecanoyl-phorbol-13-acetate, and produced more hydrogen peroxide than did the liver macrophages. Both cell types responded chemotactically to **C5a**. These results suggest that macrophages may develop specialized functions depending on the needs of their local environment. Using one and two dimensional SDS-polyacrylamide gel electrophoresis, we also compared the production of newly synthesized proteins by Kupffer cells and peritoneal macrophages. In general, the macrophages were found to produce similar types and numbers of proteins with some exceptions. These included proteins that were unique to peritoneal macrophages and other proteins observed only in Kupffer cells. The production of these proteins in liver macrophages did not appear to correlate with levels of functional activation, but may be more related to the tisuse origin of the cells.

9/7/13 (Item 13 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

6444091 BIOSIS Number: 85044612
GENERATION OF **COMPLEMENT** ANAPHYLATOXINS AND C5B-9 BY CRYSTALLINE CHOLESTEROL OXIDATION DERIVATIVES DEPENDS ON HYDROXYL GROUP NUMBER AND POSITION
SEIFERT P S; KAZATCHKINE M D
INSERM U28, HOP. BROUSSAIS, 95 RUE DIDOT, 75014 PARIS, FR.
MOL IMMUNOL 24 (12). 1987. 1303-1308. CODEN: MOIMD
Full Journal Title: Molecular Immunology
Language: ENGLISH
Cholesterol crystals activate the human alternative **complement** pathway. Loss of Factor B hemolytic activity in C2-deficient serum was comparable to that in a normal human serum after incubation with cholesterol crystals. Consumption of Factor B hemolytic activity in normal serum incubated with cholesterol occurred in a time- and dose-dependent manner. The reduced capacity of crystals-absorbed serum to activate C2, but not Factor B, on fresh crystals, indicated that cholesterol mediates **antibody** -dependent classical pathway activation in addition to alternative pathway activation in whole serum. Cholestane triol, an oxidation derivative of cholesterol which bears three hydroxyl groups, cleaved 5-fold more C3 than cholesterol in normal human serum. Three cholestrol derivatives, each bearing two hydroxyl groups, were intermediate activators between cholesterol and cholestane triol. The compounds differed, however, in their **complement**-activating ability, indicating that hydroxyl position as well as number exerts an influence on **complement** activation. Measurements of C3adesArg and C5adesArg antigens in cholesterol crystal treated serum revealed that approx. 10% of total serum C3 was cleaved and that, on a molar basis, only 3% C5 **cleavage** occurred relative to C3 **cleavage**. For 1 mole of **C5a** generated, 0.1 moles of fluid-phase C5b-9 was detected. Although the extent of C3 **cleavage** varied with each cholesterol derivative depending on the position and number of hydroxyl groups, the relative coupling efficiency of C3 and C5 **cleavage** and **C5a** and C5b-9 generation was similar for all compounds. The ability of cholesterol and its oxidation products to generate anaphylatoxins and C5b-9 complexes may be of importance in mediating inflammatory processes involved in atherogenesis.

9/7/14 (Item 1 from file: 72)
DIALOG(R)File 72:EMBASE

9970757 EMBASE No: 96145975
Cleavage of the human **C5a** receptor by proteinases derived

from Porphyromonas gingivalis: **Cleavage** of leukocyte **C5a** receptor

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Advances in Experimental Medicine and Biology (USA) , 1996, 398/- (155-164) CODEN: AEMBA ISSN: 0065-2598
LANGUAGES: English SUMMARY LANGUAGES: English

The anaerobic bacteria R gingivalis has been implicated as a primary causative agent in adult periodontitis. Several proteinases are produced by this bacteria and it is suggested that they contribute to virulence and to local tissue injury resulting from infection by P. gingivalis. Collagenases and cysteine proteinases (i.e., the gingipains) have been characterized as the predominant vesicular enzymes produced by this bacterium. It has been shown that an arginine-specific cysteine proteinase from P. gingivalis, called gingipain-1 or Arg-gingipain, can selectively cleave **complement** components C3 and C5. In the case of C5, **cleavage** by Arg-gingipain results in the generation of **C5a**, a potent chemotactic factor for PMNs. Since these bacterial proteinases are capable of generating pro-inflammatory factors at sites of infection, we examined the possibility that gingipains or other proteinases from this bacterium might attack or destroy cell surface proteins, such as receptor molecules. Using an affinity-purified rabbit **antibody** raised against residues 9-29 of the **C5a** receptor (i.e., C5aR; CD88), the signal transmitting element for the pro-inflammatory mediator **C5a** , we demonstrated that the mixture of proteinases in P. gingivalis vesicles cleaves the **C5a** receptor on human neutrophils. This vesicular proteinase activity did not require cysteine activation which indicates that proteinases other than the gingipains may be responsible for **cleavage** of the C5aR molecule. In addition, the purified Lys-gingipain, but not Arg-gingipain, also cleaved C5aR on the human neutrophils. The N-terminal region of C5aR (residues 9-29, PDYGHYDDKDTLDLNTPVDKT) was readily cleaved by chymotrypsin, but not by trypsin, despite the presence of potential trypsin (i.e., lysyl- X) **cleavage** sites. The specific sites of C5aR 9-29 peptide **cleavage** were determined by mass spectroscopy for both chymotrypsin and Lys-gingipain. These studies suggest that the proteolytic activity in the bacterial vesicles that is responsible for cleaving C5aR is primarily a non-tryptic proteinase, distinct from either Arg- or Lys-gingipain. Consequently, there appear to be additional proteinase(s) in the vesicles that attacks the cell surface molecule C5aR which are not the same (i.e., Arg- and Lys-gingipain) as were shown to generate pro-inflammatory activity from **complement** components C3 and C5. Evidence that the proteinases which attack the inflammatory precursor molecules (i.e., C3 and C5) exhibit different specificities than those that attack receptors to these bioactive **complement** products makes a particularly interesting story of how this bacteria avoids major host defense mechanisms. It is well known that generation of pro-inflammatory factors such as C3a and **C5a** at extra-vascular sites can promote edema, leukocyte recruitment and cellular activation responses that could lead to the release of toxic oxygen products and to phagocytosis of the bacteria. Destruction of receptors to these cellular activating factors generated by bacterial proteinases may eliminate the ability of these (i.e., **complement**-derived) and other mediators to carry out their anti-bacterial actions and thereby limit the host's defense mechanisms in responses to the infecting bacteria. The concept of anti-bacterial responses (i.e., oxygen radical generation and phagocytosis) being effectively eliminated at the injury site, by bacterial proteinases acting at the cellular receptor level, has not been studied in detail. In this case, the situation is particularly unusual because, once the bacterial gingipains generate potent plasma-derived inflammatory factors that can enhance edema and deliver essential nutrients to the bacteria, other bacterial proteinases may destroy their cellular receptors. These receptors transmit the signal activation mechanisms in the infiltrating cells that elicit bacterial killing. It is this series of events which might explain the ability of these anaerobes to persist and flourish in gingival tissue.

9/7/15 (Item 2 from file: 72)
DIALOG(R)File 72:EMBASE

9391150 EMBASE No: 94329738
Inactivation of human anaphylatoxin **C5a** and **C5a** des-Arg through **cleavage** by the plasminogen activator activity of a human fibrosarcoma cell line
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J. BIOL. CHEM. (USA) , 1994, 269/41 (25529-25533) CODEN: JBCHA ISSN: 0021-9258
LANGUAGES: English SUMMARY LANGUAGES: English
The HT-1080 human fibrosarcoma cell line exhibited a plasminogen-dependent ability to inactivate recombinant anaphylatoxin **C5a** or zymosan-activated serum. The inactivation was obtained at physiological levels of both plasminogen (2 microM) and **C5a** (1-5 nM). Inactivated **C5a** and zymosan-activated serum were no longer able to induce chemotaxis and degranulation of neutrophils. Inactivation of **C5a** paralleled the emergence of plasmin activity, assayed by **cleavage** of the synthetic substrate H-D-valyl-L-leucyl-L-lysine-p-nitroanilide (S-2251). Both **C5a** inactivation and S-2251 **cleavage** were inhibited by the plasmin inhibitor alpha2-antiplasmin, the urokinase inhibitor amiloride, and by anti-urokinase **antibodies**. In a cell-free system, inactivation of **C5a** was shown to depend on the simultaneous presence of urokinase and plasminogen and was inhibited by alpha2-antiplasmin and by anti- urokinase **antibodies** . SDS-polyacrylamide electrophoresis demonstrated the **cleavage** of **C5a** by the plasminogen activation system and inhibition of the **cleavage** by amiloride. Amino acid sequencing of the band corresponding to the **C5a** degradation product revealed that **C5a** was cleaved at positions Lys14- His15 and Arg10-Ile44; **cleavage** at position Arg40-Ile41 seemed to be responsible for the loss of activity. Since neoplastic cells extensively produce and exhibit plasminogen activator activity, the present observations suggest that plasminogen activation may, by inactivation of **C5a**, reduce the anti-tumor immune response and support the immunological escape phenomenon of tumors.

9/7/16 (Item 3 from file: 72)
DIALOG(R)File 72:EMBASE

7552899 EMBASE No: 89275181
Role of **C5a** in the induction of tumoricidal activity in C3H/HeJ (Lps(d)) and C3H/OuJ (Lps(n)) macrophages
Hogan M.M.; Yancey K.B.; Vogel S.N.
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J. LEUKOCYTE BIOL. (USA) , 1989, 46/6 (565-570) CODEN: JLBIE ISSN: 0741-5400
LANGUAGES: English
Thioglycollate-elicited macrophages from C3H/HeJ (Lps(d)) and C3H/OuJ (Lps(n)) mice were cultured in a two-signal, tumoricidal assay using recombinant interferon-gamma (rIFN-gamma) as the 'priming' signal and recombinant human **C5a** (rC5a) as the 'trigger' signal. These experiments were compared directly with a well established, two-signal tumoricidal assay in which rIFN-gamma was used as the 'priming' signal and protein-rich, butanol-extracted lipopolysaccharide (But-LPS) as the 'trigger' signal. These studies showed that rIFN-alpha-primed macrophages can be triggered in a dose-dependent manner by rC5a to effect high levels of tumoricidal activity. Maximum levels of cytotoxicity achieved using this

endogenously produced, biologically active peptide as a 'trigger' signal were comparable to those obtained using But-LPS. Moreover, experiments in which anti-C5 **antibody** was included in macrophage cultures stimulated with rIFN-gamma and But-LPS showed a significant reduction ($P < .05$) in tumoricidal activity. Because LPS has been shown to induce macrophage C5 production and enzyme release, these findings suggest that macrophage-derived C5 is locally converted to **C5a** (or some other biologically active C5 **cleavage** fragment), which functions as an autocrine trigger signal for the induction of tumoricidal activity. In summary, these data suggest 1) that rC5a can provide a 'second signal' to rIFN-gamma-primed murine macrophages for the induction of tumoricidal activity and 2) that macrophage-derived C5 or **C5a** may represent an autocrine signal induced by exogenous 'trigger signals.'

9/7/17 (Item 4 from file: 72)
DIALOG(R)File 72:EMBASE

7454849 EMBASE No: 89177063
Ba and Bb fragments of factor B activation: Fragment production, biological activities, neoepitope expression and quantitation in clinical samples
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COMPLEMENT INFLAMM. (Switzerland) , 1989, 6/3 (175-204) CODEN: CMPIE
ISSN: 1012-8204
LANGUAGES: English
Factor B is a centrally important component of the alternative **complement** pathway. Alternative pathway activation results in factor B **cleavage** and production of the amino-terminal Ba and the carboxyl-terminal Bb fragments which have molecular weights of approximately 30,000 and 63,000 daltons, respectively. Both Ba and Bb fragments have been reported to express a variety of biological activities in vitro. Thus, binding of Ba and Bb fragments to specific B lymphocyte surface receptors modulates proliferation of prestimulated B cells. In addition, the enzymatically active Bb fragment induces activation and spreading of human and murine macrophages and monocytes as well as regulates **C5a** des Arg chemotactic activity. The fractional catabolic rate and metabolism of factor B in vivo is similar to that of C3, C4 and C5 **complement** proteins, which are among the most metabolically active plasma proteins in the circulatory system. Factor B hyperconsumption and increased catabolism, concomitant with factor B fragment production, occurs in a wide variety of diseases, including gram-negative sepsis, autoimmune diseases and burns. Measurement of alternative pathway activation in vivo has been attempted utilized a number of different techniques to quantitate factor B fragments in biological fluids. However, the recent development of enzyme immunoassays (EIA) employing monoclonal **antibodies** (MoAbs) reactive with factor B fragment neoepitopes provides the best approach currently available for the quantitation of factor B activation fragments. Results obtained using these new MoAb-based EIAs have indicated that factor B fragment concentrations were elevated, as compared with normal donor levels, in EDTA plasma samples obtained from patients with rheumatoid arthritis and systemic lupus erythematosus (SLE). Plasma concentrations of factor B fragments, especially Ba fragment levels, in these patients showed a positive correlation with disease activity scores. One of the highest disease activity correlations was obtained with Ba fragment measurements in SLE plasma samples. In fact, the results strongly suggested that quantitation of Ba fragment levels in SLE plasma samples more accurately reflected disease activity and was a more sensitive predictor of impending flare in these patients than any other test(s) currently available.

9/7/18 (Item 5 from file: 72)
DIALOG(R)File 72:EMBASE

7172278 EMBASE No: 88171394

Molecular organization and function of the **complement** system

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ANNU. REV. BIOCHEM. (USA) , 1988, 57/- (321-347) CODEN: ARBOA ISSN: 0066-4154

LANGUAGES: English

Complement plays an important role in host defense against infectious agents and in the inflammatory process. It consists of 20 plasma proteins that function either as enzymes or as binding proteins. The wider **complement** system includes multiple distinct cell-surface receptors that exhibit specificity for physiological fragments of **complement** proteins and that occur on inflammatory cells and cells of the immune system. In addition, there are regulatory membrane proteins that prevent autologous **complement** activation and protect host cells from accidental **complement** attack. The system is organized in two activation pathways, designated 'classical' and 'alternative', and in the cytolytic pathway of membrane attack. Both activation pathways contain an initial enzyme that catalyzes the formation of the target-cell-bound C3 convertase which in turn generates the C5 convertase. Binding of **antibody** molecules to a foreign particle results in activation of the classical pathway, which is **antibody** -dependent. In contrast, the alternative pathway does not require **antibody** for its activation. It exists in an activated state at all times due to the spontaneous reaction of its major component, C3, with water. Native C3 is endowed with an internal thioester that undergoes hydrolysis at a slow rate giving rise to a functionally active C3 molecule, C3(H2O). When, as a result of formation of the initial enzyme, native C3 is cleaved and the fragment C3b is deposited on the surface of particles, the alternative pathway is enabled to distinguish between self and nonself. Only on particles recognized as foreign will amplification of C3b deposition occur and membrane attack be initiated. Both activation pathways eventuate in proteolytic **cleavage** of the protein C5 and thus in assembly of the membrane attack complex (MAC) from five hydrophilic precursor proteins. Through its metastable membrane-binding site, the forming MAC binds firmly to target membranes owing to hydrophobic interaction with the lipid bilayer. The final events of MAC assembly are unfolding and polymerization of the protein C9 within the target membrane, which cause weakening of membrane structure and formation of transmembrane channels. Cytotoxic lymphocytes kill their target cells using a protein that resembles in some respects C9. It undergoes polymerization in target membranes to form transmembrane channels and it shows an immunochemical relationship to C9. Whereas this C9-related protein (C9RP) is constitutive in natural killer (NK) cells, it is newly synthesized upon activation of resting cytotoxic T lymphocytes (CTL). C3 is pivotal in the organization and function of the **complement** system. It is the precursor of biologically active fragments that function by associating with other **complement** proteins or by binding to cell-surface receptors. C3 harbors at least 10 distinct binding sites, one of which, the thioester, enables the molecule to bind covalently to target cells and particles such as immune complexes. The cellular receptors for fragments of C3 have assumed increasing importance. One has been identified with the Epstein-Barr virus receptor on B lymphocytes; two others have been shown to belong structurally to the LFA-1 family of leukocyte surface-adhesive molecules. The anaphylotoxins are proteolytic activation peptides of the proteins C3, C4, and C5. These peptides are hormonelike messengers that bind to specific receptors of neutrophils, monocytes, macrophages, mast cells, and smooth muscle cells to elicit a variety of cellular responses. Particularly one of these peptides, **C5a**, is a highly potent mediator of inflammation, causing chemotactic migration, cell adhesion, release of hydrolytic enzymes, and the formation of arachidonic acid metabolites and active oxygen species. The large number of seemingly different proteins composing this complex biological system may appear

bewildering. Recent genetic and structural analyses have uncovered the existence of remarkable relationships among these proteins. Four families of proteins can readily be discerned, and these will be described in the next section. The description of the proteins will be followed by an illumination of the functional versatility of C3 and a discussion of molecular mechanisms pertaining to recognition, activation, and membrane attack. The chapter will conclude by pointing out that the molecular basis of lymphocyte cytotoxicity is closely related to the cytolytic mechanism of **complement**.

9/7/19 (Item 6 from file: 72)
DIALOG(R)File 72:EMBASE

6424478 EMBASE No: 87161185
IgG binding to cytoskeletal intermediate filaments activates the **complement** cascade
Hansson G.K.; Lagerstedt E.; Bengtsson A.; Heideman M.
Department of Clinical Chemistry, Gothenburg University, Sahlgrenska Sjukhuset, S 413 45 Gothenburg SWEDEN
EXP. CELL RES. (USA) , 1987, 170/2 (338-350) CODEN: ECREA
LANGUAGES: ENGLISH
The cellular plasma membrane becomes permeable to macromolecules during the cell injury process. This results in exposure of the interior of the cell to plasma proteins and to high-affinity binding of the Fc part of IgG to intermediate filaments (Hansson, G.K., Starkebaum, G.A., Benditt, E.P. & Schwartz S.M., Proc Natl Acad Sci USA 81 (1984) 3103). Such IgG binding could be an early step in a process that serves to eliminate the injured cell. We now have identified its effect on the **complement** system. Intermediate filaments were reconstituted in vitro from purified vimentin, and incubated with plasma proteins. Cross-linker experiments showed binding of the heavy chain of IgG to vimentin, indicating that the vimentin protein carries an Fc-binding site. In contrast, no direct binding of **complement** factor Clq to vimentin could be detected. Binding of both IgG and Clq could, however, be detected by immunofluorescence when cytoskeletons of cultured endothelial cells were incubated with fresh serum. Therefore, IgG binding to filaments in the presence of serum is accompanied by Clq binding to IgG. This was in turn followed by fixation of C4 and C3 to intermediate filaments in a process that was dependent on both Casup 2sup +, Mgsup 2sup + and Clq, indicating that it was part of a **complement** activation via the classical pathway. Exposure of fresh serum to intermediate filaments also resulted in production of the anaphylatoxic **complement cleavage** fragment, C3a, with a dose-response relationship between the amount of filaments present and the amount of C3a generated. Chemotactic activity towards granulocytes and monocytes was also generated by exposure of serum to intermediate filaments, and this activity was dependent on the presence of **complement** factor C5 and on the classical **complement** activation cascade, implying that it was due to the **C5a** peptide. Exposure of the interior of the cell to plasma proteins thus results in binding of IgG to intermediate filaments and activation of the **complement** cascade via the classical pathway. This, in turn generates bioactive mediators which may recruit leukocytes to the injured cell (**C5a**) and have profound effects on vascular permeability (C3a, **C5a**). We propose that this is part of a scavenger mechanism for the elimination of damaged cells.

9/7/20 (Item 7 from file: 72)
DIALOG(R)File 72:EMBASE

6393679 EMBASE No: 87130339
An in vitro model of the wound microenvironment: Local phagocytic cell abnormalities associated with in situ **complement** activation

Yamada Y.; Hefter K.; Burke J.F.; Gelfand J.A.
Department of Surgery, Massachusetts General Hospital, Boston, MA USA
J. INFECT. DIS. (USA) , 1987, 155/5 (998-1004) CODEN: JIDIA
LANGUAGES: ENGLISH

An in vitro model was developed to investigate the inflammatory response to tissue damage. Human fibroblasts were heat killed and incubated with serum. **Complement** studies showed activation of the alternative pathway proportional to the number of dead cells; C3 was fixed on dead cells, and **C5a** was generated. Neutrophils (PMNLs) adhered to killed fibroblasts, a process requiring fresh serum. After adhering to killed fibroblasts in the presence of serum, PMNLs exhibited depressed chemotactic responsiveness to activated serum and reduced bactericidal activity against preopsonized Staphylococcus aureus. These data suggest that thermally killed cells activate and fix **complement**, a process generating **cleavage** products that, in turn, recruit PMNLs and bind them to the inflammatory site. Thus, in our model, dead tissue activates humoral mechanisms and inflammatory cells; this process results in depressed in situ host-defense function upon subseqent local challenge with microbes.

9/7/21 (Item 8 from file: 72)
DIALOG(R)File 72:EMBASE

6141170 EMBASE No: 86136230
Complement mediated inhibition of immune precipitation and solubilization generate different concentrations of **complement** anaphylatoxins (C4a, C3a, **C5a**)
Schifferli J.A.; Steiger G.; Paccaud J.-P.
Clinique Medicale, Hopital Cantonal Universitaire, 1211 Geneva 4 SWITZERLAND
CLIN. EXP. IMMUNOL. (ENGLAND) , 1986, 64/2 (407-414) CODEN: CEXIA
LANGUAGES: ENGLISH

Complement prevents the formation of insoluble immune complexes (inhibition of immune precipitation (IIP)), and solubilizes preformed immune aggregates (solubilization (SOL)). Since the mechanism of **complement** activation differs in these two reactions, it is possible that they differ also in the amount of **complement** fragments released, in particular the anaphylatoxins C3a, **C5a** and C4a. We measured C4 and C3 consumption, and the formation of **complement** anaphylatoxins during IIP and SOL using two different immune complex models (BSA, rabbit anti-BSA; tetanus toxoid (TT), human anti-TT). At equal immune complex concentrations in both models, SOL was more efficient than IIP at cleaving C3, and more C3a and **C5a** was released. Comparing the two reactions, C3a formation was followed by more C5 **cleavage** (**C5a**) during SOL. Similarly C4a formation (classical pathway activation) was followed by more C3 **cleavage** (C3a: classical and alternative pathway activations), during SOL. It is suggested that in vivo SOL of insoluble complexes is rapidly accompanied by a damaging phlogistic reaction, whereas IIP produces less inflammation.

9/7/22 (Item 9 from file: 72)
DIALOG(R)File 72:EMBASE

5955139 EMBASE No: 85200649
Effect of **C5a** on isolated guinea pig atria
Regal J.F.
Department of Pharmacology, University of Minnesota-Duluth, Duluth, MN 55812 USA
IMMUNOPHARMACOLOGY (USA) , 1985, 9/1 (27-31) CODEN: IMMUD
LANGUAGES: ENGLISH

Cleavage of the **complement** protein C5 by activation of the **complement** system yields a low molecular weight fragment, **C5a**.

Previous reports of other researchers indicate that among the biological activities of **C5a** is an ability to alter cardiac function. However, these studies have varying results. The goal of the present study was thus to determine both the chronotropic and inotropic effects of guinea pig **C5a** and the tachyphylaxis to guinea pig **C5a** in isolated atria of the guinea pig. Isolated right atria respond to guinea pig **C5a** with a consistent concentration-related positive inotropic and chronotropic response. An inotropic response to guinea pig **C5a** was seen in both spontaneously beating right atria and paced left atria. The inotropic and chronotropic responses to guinea pig **C5a** in the right atria were clearly tachyphylactic. Studies using the Hsub 2 receptor antagonist metiamide indicate that the positive chronotropic response to guinea pig **C5a** is at least in part a histamine-mediated response. Further studies are required to determine whether the conflicting results in various studies are due to the use of **C5a** from various species.

9/7/23 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

06357172 90171540
The Ba fragment of **complement** factor B inhibits human B lymphocyte proliferation.
Ambrus JL Jr; Peters MG; Fauci AS; Brown EJ
Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.
J Immunol (UNITED STATES) Mar 1 1990, 144 (5) p1549-53, ISSN 0022-1767 Journal Code: IFB
Contract/Grant No.: AI 24674, AI, NIAID
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Normal human B lymphocyte function is finely regulated by both positive and negative signals at each stage of activation, proliferation, and differentiation. Activation signals include antigen and surface Ig cross-linking agents such as anti-mu or anti-delta. Signals inducing proliferation include IL-2, high m.w.-B cell growth factor (BCGF), and low m.w.-BCGF. IL-2 as well as IL-6 and other partially characterized B cell differentiation factors can induce terminal differentiation of proliferating B cells into Ig-secreting plasma cells. Various C components have been described to regulate B cell function including Bb that enhances proliferation, **C5a** that enhances Ig production, and C3a that inhibits Ig production. In our study, we examined the ability of the factor B **cleavage** fragment Ba to influence human B cell function. Ba did not affect the activation of resting B cells but inhibited the proliferation of activated B cells stimulated with either high m.w.-BCGF or low m.w.-BCGF. The inhibition occurred with doses of Ba as low as 1 microgram/ml (29 nM). Ba was found to bind to activated human B lymphocytes in a saturable manner with an apparent K of approximately 25 nM and an apparent Bmax of 56,000 sites/cell. A peptide made of the carboxy terminal 10 amino acids of Ba (GHGPGEQQKR), was also found to inhibit growth factor induced proliferation of activated B cells but at an ID50 of approximately 5 microM. Finally, Ba was found to inhibit the terminal differentiation of Staphylococcus aweus Cowan-activated B cells stimulated with B cell differentiation factors but not Ig secretion by the partially differentiated EBV-transformed cell line SKW.6. Thus, concentrations of Ba achievable in vivo at sites of active inflammation were found to act on human B lymphocytes by inhibiting their proliferation. This may act to limit the immune response to a specific antigenic challenge.

9/7/24 (Item 2 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

06136798 86294263
The biology and pathophysiology of **complement** receptors.
Lambris JD; Tsokos GC
Anticancer Res (GREECE) May-Jun 1986, 6 (3 Pt B) p515-23, ISSN 0250-7005 Journal Code: 59L
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW
Activation of the **complement** cascade leads to the generation of multiple breakdown products which bind on to specific cellular receptors and regulate their function. In this review, we describe the biochemical and physiological features of the 7 known **complement** receptors. Four of them (**complement** receptors 1, 2, and 3 and receptors for C3a) bind **cleavage** fragments of the third component of the **complement** and three have specificity for C1q, factor H and **C5a**. In patients with systemic lupus erythematosus, a unique human autoimmune disorder, the numbers of CR1 on the surface of the red blood cells are decreased; in this review we discuss the implications in the pathogenesis of SLE. A number of patients have now been reported whose cells lack CR3 from their surface; this deficiency is associated with a number of immune cell dysfunctions which are discussed in detail. Finally, we discuss aberrations in the expression of **complement** receptors in certain human leukemic cells. (119 Refs.)

9/7/25 (Item 3 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

05864796 89163902
The human C3b receptor (CR1).
Weiss L; Fischer E; Haeffner-Cavaillon N; Jouvin MH; Appay MD; Bariety J; Kazatchkine M
Unite d'Immunopathologie, Hopital Broussais, Paris, France.
Adv Nephrol Necker Hosp (UNITED STATES) 1989, 18 p249-69, ISSN 0084-5957 Journal Code: 2NV
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
The human **complement** system is comprised of 19 plasma components and regulatory proteins and of at least 9 distinct cellular receptors for these proteins or their activation fragments. The important role of **complement** in host defense against infection is related to its capacity to opsonize microorganisms, lyze target cells, and induce the release of inflammatory mediators from leukocytes. **Complement** participates in the processing and clearance of immune complexes and in regulation of the immune response. Most of the biologic effects derived from **complement** activation depend on ligand-receptor interactions between **complement** proteins or their **cleavage** fragments and specific receptors on cells. Two types of ligands are generated during **complement** activation: soluble low-molecular-weight ligands, such as the anaphylatoxins C3a and **C5a**, and so-called bifunctional ligands that attach both to the target of **complement** activation (opsonins) and to the appropriate receptor on effector cells. The most abundant **complement** protein in plasma is C3. Activation of the classic and alternative **complement** pathways generates C3 convertases that cleave C3 into an anaphylatoxic fragment, C3a, and a major fragment, C3b, which is capable of forming a covalent linkage with the targets of **complement** activation. Surface-bound C3b is the preferential ligand for the C3b receptor, CR1 (CD 35), which is expressed on most peripheral blood cells. The receptor plays an important role in the processing of immune complexes, the phagocytosis of C3b-bearing microorganisms, and regulation of the immune response. The cellular expression of the molecule is decreased in patients with systemic lupus erythematosus (SLE) and in patients infected with the human immunodeficiency virus (HIV). (121 Refs.)

9/7/26 (Item 4 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

04949060 86276678

The activation of C5 in the fluid phase and in the absence of C3 through the classical pathway of the **complement** system.

Kitamura H; Tsuboi M; Nagaki K

Immunology (ENGLAND) Jul 1986, 58 (3) p459-65, ISSN 0019-2805
Journal Code: GH7
Languages: ENGLISH
Document type: JOURNAL ARTICLE

Unsensitized guinea-pig erythrocytes (Egp) were lysed by a combination of eight isolated, human-derived **complement** components, Cls, C4, C2, C5, C6, C7, C8 and C9 (Cls-C9exC3), even in the presence of anti-C3. It was determined that a factor was generated in the reaction mixture of Cls, C4, C2, C5 and C6, which had a lytic activity against Egp when C7, C8 and C9 were added. The lytic factor was similar to C56 in the following properties: the activity of the lytic factor decreased when incubated with C7 prior to its reaction with Egp, the lytic factor did not bind to Egp by itself but it did bind in the presence of C7, EDTA did not have any inhibitory effect on the lytic factor, and the activity of the lytic factor was lost by treatment with anti-C5 or anti-C6 but not by treatment with anti-C4. Furthermore, **C5a**, a **cleavage** product of C5, was clearly detected in the reaction mixture of Cls, C4, C2 and C5. These findings indicate that C5 can be activated proteolytically into **C5a** and C5b in the fluid phase solely by the classical pathway C3 convertase, C42, without any participation of C3.

9/7/27 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

93005893 CA: 93(1)5893y JOURNAL

Mediators of immune complex-induced aggregation of polymorphonuclear neutrophils. I. C5a anaphylatoxin, neutrophil cationic proteins and their cleavage fragments

AUTHOR(S): Camussi, G.; Tetta, C.; Bussolino, F.; Caligaris Cappio, F.; Coda, R.; Masera, C.; Segoloni, G.
LOCATION: Osp. Maggiore S. G. Battista, Univ. Torino, Turin, Italy

JOURNAL: Int. Arch. Allergy Appl. Immunol. DATE: 1980 VOLUME: 62
NUMBER: 1 PAGES: 1-15 CODEN: IAAAAM ISSN: 0020-5915 LANGUAGE: English
SECTION:
CA015013 Immunochemistry
IDENTIFIERS: neutrophil aggregation mediator immune complex, complement C5a peptide neutrophil aggregation
DESCRIPTORS:
Neutrophil...
aggregation of, complement C5a anaphylatoxin and cationic proteins and fragments effect on
Antibodies...
immune complexes, neutrophil aggregation in response to anaphylatoxin C5a and cationic proteins in relation to
Anaphylatoxins,C5a... Complement,C5a...
neutrophil aggregation in response to, immune complex formation in relation to
Proteins,cationic...
of neutrophils, neutrophil aggregation induced by immune complex interaction in response to
CAS REGISTRY NUMBERS:
7439-95-4 7440-70-2 biological studies, neutrophil aggregation induced by immune complexes in response to

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Ref    Items  Index-term
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E2         6  AU=ROLLINS, SCOTT
E3         0 *AU=ROLLINS, SCOTT ?
E4        35  AU=ROLLINS, SCOTT A.
E5         1  AU=ROLLINS, SCOTT ALAN
E6         5  AU=ROLLINS, SEAN M.
E7         1  AU=ROLLINS, STEPHEN
E8         1  AU=ROLLINS, T. E.
E9         1  AU=ROLLINS, THOMAS
E10       12  AU=ROLLINS, THOMAS E.
E11        1  AU=ROLLINS, TREVEN
E12       10  AU=ROLLINS, V.

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                1  AU=ROLLINS, SCOTT ALAN
     S10       42  E2-E5
? s s10 and complement

               42  S10
           121257  COMPLEMENT
     S11       35  S10 AND COMPLEMENT
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>>>Records from unsupported files will be retained in the RD set.
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     S12       34  RD S11 (unique items)
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 12/3/1     (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 1998 American Chemical Society. All rts. reserv.

  127120618    CA: 127(9)120618v    CONFERENCE PROCEEDING
  Amelioration of lupuslike autoimmune disease in NZB/W F1 mice after
treatment with a blocking monoclonal antibody specific for complement
component C5
  AUTHOR(S): Wang, Yi; Hu, Qile; Madri, Joseph A.; Rollins, Scott A.;
Chodera, Amy; Matis, Louis A.
  LOCATION: Alexion Pharmaceuticals, 25 Science Park, New Haven, CT, 06511,
USA
  JOURNAL: Controlling Complement Syst. Novel Drug Dev., (IBC Conf.)
  EDITOR: Mazarakis, Helen (Ed), Swart, Sarah Jane (Ed),  DATE: 1997
  PAGES: 89-109  CODEN: 64QOAM  LANGUAGE: English  MEETING DATE: 19960000
  PUBLISHER: International Business Communications,Southborough, Mass


 12/3/2     (Item 2 from file: 399)
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DIALOG(R)File 399:CA SEARCH(R)

127079964 CA: 127(6)79964q JOURNAL
Inhibition of complement activity by humanized anti-C5 antibody and single-chain Fv
AUTHOR(S): Thomas, Thomas C.; Rollins, Scott A.; Rother, Russell P.; Giannoni, Michelle A.; Hartman, Sandra L.; Elliott, Eileen A.; Nye, Steven H.; Matis, Louis A.; Squinto, Stephen P.; Evans, Mark J.
LOCATION: Alexion Pharmaceuticals, New Haven, CT, 06511, USA
JOURNAL: Mol. Immunol. DATE: 1997 VOLUME: 33 NUMBER: 17/18 PAGES: 1389-1401 CODEN: MOIMD5 ISSN: 0161-5890 PUBLISHER ITEM IDENTIFIER: 0161-5890(96)00078-8 LANGUAGE: English MEETING DATE: 19960000
PUBLISHER: Elsevier

12/3/3 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125165528 CA: 125(13)165528r JOURNAL
Amelioration of lupus-like autoimmune disease in NZB/W F1 mice after treatment with a blocking monoclonal antibody specific for complement component C5
AUTHOR(S): Wang, Yi; Hu, Qile; Madri, Joseph A.; Rollins, Scott A.; Chodera, Amy; Matis, Louis A.
LOCATION: Immumobiology Program, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1996 VOLUME: 93 NUMBER: 16 PAGES: 8563-8568 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English

12/3/4 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125084172 CA: 125(7)84172t JOURNAL
Expression of human CD59 in transgenic pig organs enhances organ survival in an ex vivo xenogeneic perfusion model
AUTHOR(S): Kroshus, Timothy J.; Bolman, R. Morton, III; Dalmasso, Agustin P.; Rollins, Scott A.; Guilmette, Edward R.; Williams, Barry L.; Squinto, Stephen P.; Fodor, William L.
LOCATION: Veterans Affairs Medical Center, University Minnesota, Minneapolis, MN, 55455, USA
JOURNAL: Transplantation DATE: 1996 VOLUME: 61 NUMBER: 10 PAGES: 1513-1521 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

12/3/5 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124325364 CA: 124(24)325364u PATENT
Retroviral transduction of cells using soluble complement inhibitors
INVENTOR(AUTHOR): Rother, Russell P.; Rollins, Scott A.; Mason, James M.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9603146 A1 DATE: 960208
APPLICATION: WO 95US8924 (950714) *US 278550 (940721)
PAGES: 49 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A
DESIGNATED COUNTRIES: AU; CA; JP DESIGNATED REGIONAL: AT; BE; CH; DE; DK ; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

12/3/6 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124143157 CA: 124(11)143157w JOURNAL
Monoclonal antibodies directed against human C5 and C8 block complement-mediated damage of xenogeneic cells and organs
AUTHOR(S): Rollins, Scott A.; Matis, Louis A.; Springhorn, Jeremy P.; Setter, Eva; Wolff, Dennis W.
LOCATION: Department of Immunobiology, Alexion Pharmaceuticals, Inc., New haven, CT, 06511, USA
JOURNAL: Transplantation DATE: 1995 VOLUME: 60 NUMBER: 11 PAGES: 1284-92 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

12/3/7 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124143156 CA: 124(11)143156v JOURNAL
Complement inhibition with an anti-C5 monoclonal antibody prevents acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation
AUTHOR(S): Kroshus, Timothy J.; Rollins, Scott A.; Dalmasso, Agustin P.; Elliott, Eileen A.; Matis, Louis A.; Squinto, Stephen P.; Bolman, R. Morton, III
LOCATION: Department of Surgery, University of Minnesota, Minneapolis, MN , USA
JOURNAL: Transplantation DATE: 1995 VOLUME: 60 NUMBER: 11 PAGES: 1194-202 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

12/3/8 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124127101 CA: 124(10)127101t PATENT
Anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
INVENTOR(AUTHOR): Evans, Mark J.; Matis, Louis; Mueller, Eileen Elliott; Nye, Steven H.; Rollins, Scott; Rother, Russell P.; Springhorn, Jeremy P.; Squinto, Stephen P.; Thomas, Thomas C.; et al.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9529697 A1 DATE: 951109
APPLICATION: WO 95US5688 (950501) *US 236208 (940502)
PAGES: 159 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-038/36A; A61K-039/00B; A61K-039/395B; C07K-014/00B; C07K-014/75B; C07K-016/00B; C07K-016/18B; C07K-016/36B; C07K-016/46B; C12N-005/10B; C12N-005/20B; C12N-015/09B; C12N-015/10B; C12N-015/13B; C12N-015/63B; C12P-021/02B; C12P-021/08B DESIGNATED COUNTRIES: AM; AU; BB; BG; BR; BY; CA; CN; CZ; EE; FI; GE; HU; IS; JP; KG; KP; KR; KZ; LK; LR; LT; LV; MD; MG; MN; MX; NO; NZ; PL; RO; RU; SG; SI; SK; TJ; TM; TT; UA; UG; US; UZ; VN
DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG

12/3/9 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124114995 CA: 124(9)114995n JOURNAL
In vitro and in vivo inhibition of complement activity by a single-chain

Fv fragment recognizing human C5
AUTHOR(S): Evans, Mark J.; Rollins, Scott A.; Wolff, Dennis W.; Rother, Russell P.; Norin, Allen J.; Therrien, Denise M.; Grijalva, Galo A.; Mueller, John P.; Nye, Steven H.; et al.
LOCATION: Dep. of Mol. Development, Alexion Pharmaceuticals, New Haven, CT, 06511, USA
JOURNAL: Mol. Immunol. DATE: 1995 VOLUME: 32 NUMBER: 16 PAGES: 1183-95 CODEN: MOIMD5 ISSN: 0161-5890 LANGUAGE: English

12/3/10 (Item 10 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124028050 CA: 124(3)28050u PATENT
Chimeric complement inhibitor proteins
INVENTOR(AUTHOR): Fodor, William L.; Rollins, Scott; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9523856 A1 DATE: 950908
APPLICATION: WO 95US2945 (950301) *US 205508 (940303)
PAGES: 86 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/00A; C07K-014/00B; C07H-021/00B DESIGNATED COUNTRIES: AU; BR; CA; CN; HU; JP; KR; MX; NO; NZ; RU DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR ; IE; IT; LU; MC; NL; PT; SE

12/3/11 (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124006626 CA: 124(1)6626j JOURNAL
Enzymic remodelling of the carbohydrate surface of a xenogenic cell substantially reduces human antibody binding and complement-mediated cytolysis
AUTHOR(S): Sandrin, Mauro S.; Fodor, William L.; Mouhtouris, Effie; Osman, Narin; Cohney, Shlomo; Rollins, Scott A.; Guilmette, Edward R.; Setter, Eva; Squinto, Stephen P.; et al.
LOCATION: Molecular Immunogenetics Lab., Austin Research Inst., Heidelberg, 3084, Australia
JOURNAL: Nat. Med. (N. Y.) DATE: 1995 VOLUME: 1 NUMBER: 12 PAGES: 1261-7 CODEN: NAMEFI ISSN: 1078-8956 LANGUAGE: English

12/3/12 (Item 12 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123337462 CA: 123(25)337462s PATENT
Method for reducing immune and hemostatic dysfunctions during extracorporeal circulation
INVENTOR(AUTHOR): Rollins, Scott A.; Smith, Brian R.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.; Yale University
PATENT: PCT International ; WO 9525540 A1 DATE: 950928
APPLICATION: WO 95US3614 (950322) *US 217391 (940323)
PAGES: 34 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/00A; A61K-039/395B; C07K-016/00B; C07K-016/18B DESIGNATED COUNTRIES: AU; CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

12/3/13 (Item 13 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123312243 CA: 123(23)312243h PATENT
Recombinant preparation of terminal complement inhibitor fusion proteins lacking glycosyl-phosphatidylinositol (GPI) anchor and their use in organ transplantation
INVENTOR(AUTHOR): Rother, Russell P.; Rollins, Scott; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9523512 A1 DATE: 950908
APPLICATION: WO 95US2944 (950301) *US 205720 (940303)
PAGES: 85 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A01N-063/00A; A61K-035/14B; A61K-038/00B; C07H-017/00B; C07K-014/00B; C12N-001/00B; C12N-005/00B; C12N-005/06B; C12N-005/22B; C12N-007/01B; C12N-015/00B; C12N-015/03B; C12N-015/09B; C12N-015/06B; C12N-015/11B; C12P-100/00B; C12P-210/06B DESIGNATED COUNTRIES: AU; CA; JP DESIGNATED REGIONAL: AT; BE ; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

12/3/14 (Item 14 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123283252 CA: 123(21)283252c JOURNAL
A novel bifunctional chimeric complement inhibitor that regulates C3 convertase and formation of the membrane attack complex
AUTHOR(S): Fodor, William L.; Rollins, Scott A.; Guilmette, Edward R.; Setter, Eva; Squinto, Stephen P.
LOCATION: Dep. Mol. Dev., Alexion Pharm., Inc., New Haven, CT, 06511, USA
JOURNAL: J. Immunol. DATE: 1995 VOLUME: 155 NUMBER: 9 PAGES: 4135-8
CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English

12/3/15 (Item 15 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123283240 CA: 123(21)283240x JOURNAL
A novel mechanism of retrovirus inactivation in human serum mediated by anti-.alpha.-galactosyl natural antibody
AUTHOR(S): Rother, Russell P.; Fodor, William L.; Springhorn, Jeremy P.; Birks, Carl W.; Setter, Eva; Sandrin, Mauro S.; Squinto, Stephen P.; Rollins, Scott A.
LOCATION: Departments Molecular Development Immunobiol., Alexion Pharmaceuticals, New Haven, CT, 06511, USA
JOURNAL: J. Exp. Med. DATE: 1995 VOLUME: 182 NUMBER: 5 PAGES: 1345-55
CODEN: JEMEAV ISSN: 0022-1007 LANGUAGE: English

12/3/16 (Item 16 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123245826 CA: 123(19)245826k JOURNAL
Complement-specific antibodies: designing novel anti-inflammatories
AUTHOR(S): Matis, Louis A.; Rollins, Scott A.
LOCATION: Immunobiol. Prog., Alexion Pharm., Inc., New Haven, CT, 06511, USA
JOURNAL: Nat. Med. (N. Y.) DATE: 1995 VOLUME: 1 NUMBER: 8 PAGES: 839-42 CODEN: NAMEFI ISSN: 1078-8956 LANGUAGE: English

12/3/17 (Item 17 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123225490 CA: 123(17)225490t JOURNAL
Blockade of C5a and C5b-9 generation inhibits leukocyte and platelet activation during extracorporeal circulation
AUTHOR(S): Rinder, Christine S.; Rinder, Henry M.; Smith, Brian R.; Fitch, Jane C. K.; Smith, Michael J.; Tracey, Jayne B.; Matis, Louis A.; Squinto, Stephen P.; Rollins, Scott A.
LOCATION: Dep. of Laboratory Medicine and Anesthesiology, Yale Univ. Sch. of Medicine and Yale-New Haven Hospital, New Haven, CT, 06510, USA
JOURNAL: J. Clin. Invest. DATE: 1995 VOLUME: 96 NUMBER: 3 PAGES: 1564-72 CODEN: JCINAO ISSN: 0021-9738 LANGUAGE: English

12/3/18 (Item 18 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123196481 CA: 123(15)196481h JOURNAL
Anti-C5 monoclonal antibody therapy prevents collagen-induced arthritis and ameliorates established disease
AUTHOR(S): Wang, Yi; Rollins, Scott A.; Madri, Joseph A.; Matis, Louis A.
LOCATION: Immunobiol. Program, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1995 VOLUME: 92 NUMBER: 19 PAGES: 8955-9 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English

12/3/19 (Item 19 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123141260 CA: 123(11)141260e JOURNAL
Rapid expression of an anti-human C5 chimeric Fab utilizing a vector that replicates in COS and 293 cells
AUTHOR(S): Evans, Mark J.; Hartman, Sandra L.; Wolff, Dennis W.; Rollins, Scott A.; Squinto, Stephen P.
LOCATION: Department of Molecular Development, Alexion Pharmaceuticals, Inc., 25 Science Park, New Haven, USA
JOURNAL: J. Immunol. Methods DATE: 1995 VOLUME: 184 NUMBER: 1 PAGES: 123-38 CODEN: JIMMBG ISSN: 0022-1759 LANGUAGE: English

12/3/20 (Item 20 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123081609 CA: 123(7)81609p PATENT
Complement inhibitor proteins of non-human primates
INVENTOR(AUTHOR): Fodor, William L.; Rollins, Scott A.; Rother, Russel P.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9504756 A1 DATE: 950216
APPLICATION: WO 94US9046 (940810) *US 105735 (930811)
PAGES: 125 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07K-014/435A; C07K-014/705B; A61K-038/17B; C12N-015/12B; C12N-015/79B
DESIGNATED COUNTRIES: JP DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

12/3/21 (Item 21 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123007584 CA: 123(1)7584k JOURNAL

The complement control protein homolog of herpesvirus saimiri regulates serum complement by inhibiting C3 convertase activity
AUTHOR(S): Fodor, William L.; Rollins, Scott A.; Bianco-Caron, Stella; Rother, Russell P.; Guilmette, Edward R.; Burton, Willis V.; Albrecht, Jens-Christian; Fleckenstein, Bernhard; Squinto, Stephen P.
LOCATION: Alexion Pharmaceuticals Inc., New Haven, CT, 06511, USA
JOURNAL: J. Virol. DATE: 1995 VOLUME: 69 NUMBER: 6 PAGES: 3889-92
CODEN: JOVIAM ISSN: 0022-538X LANGUAGE: English

12/3/22 (Item 22 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122263065 CA: 122(21)263065v JOURNAL
Primate terminal complement inhibitor homologs of human CD59
AUTHOR(S): Fodor, William L.; Rollins, Scott A.; Bianco-Caron, Stella; Burton, Willis V.; Guilmette, Edward R.; Rother, Russell P.; Zavoico, George B.; Squinto, Stephen P.
LOCATION: Alexion Pharmaceuticals, Inc., New Haven, CT, 06511-1968, USA
JOURNAL: Immunogenetics DATE: 1995 VOLUME: 41 NUMBER: 1 PAGES: 51
CODEN: IMNGBK ISSN: 0093-7711 LANGUAGE: English

12/3/23 (Item 23 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122158376 CA: 122(13)158376z JOURNAL
Expression of recombinant transmembrane CD59 in paroxysmal nocturnal hemoglobinuria B cells confers resistance to human complement
AUTHOR(S): Rother, Russell P.; Rollins, Scott A.; Mennone, John; Chodera, Amy; Fidel, Seth A.; Bessler, Monica; Hillmen, Peter; Squinto, Stephen P.
LOCATION: Alexion Pharmaceuticals Inc., New Haven, CT, USA
JOURNAL: Blood DATE: 1994 VOLUME: 84 NUMBER: 8 PAGES: 2604-11
CODEN: BLOOAW ISSN: 0006-4971 LANGUAGE: English

12/3/24 (Item 24 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121298801 CA: 121(25)298801p JOURNAL
Expression of a functional human complement inhibitor in a transgenic pig as a model for the prevention of xenogeneic hyperacute organ rejection
AUTHOR(S): Fodor, William L.; Williams, Barry L.; Matis, Louis A.; Madri, Joseph A.; Rollins, Scott A.; Knight, James W.; Velander, William; Squinto, Stephen P.
LOCATION: Alexion Pharm. Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1994 VOLUME: 91
NUMBER: 23 PAGES: 11153-7 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English

12/3/25 (Item 25 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121131942 CA: 121(11)131942y JOURNAL
Protection of porcine aortic endothelial cells from complement-mediated cell lysis and activation by recombinant human CD59
AUTHOR(S): Kennedy, Scott P.; Rollins, Scott A.; Burton, Willis V.; Sims, Peter J.; Bothwell, Alfred L. M.; Squinto, Stephen P.; Zavoico, George B.
LOCATION: Dep. Vasc. Biol., Alexion Pharm. Inc., New Haven, CT, 06511, USA

JOURNAL: Transplantation DATE: 1994 VOLUME: 57 NUMBER: 10 PAGES: 1494-501 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

12/3/26 (Item 26 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120101475 CA: 120(9)101475k JOURNAL
Inhibition of complement-mediated cytolysis by the terminal complement inhibitor of herpesvirus saimiri
AUTHOR(S): Rother, Russell P.; Rollins, Scott A.; Fodor, William L.; Albrecht, Jens C.; Setter, Eva; Fleckenstein, Bernhard; Squinto, Stephen P.
LOCATION: Alexion Pharm. Inc., New Haven, CT, 06511, USA
JOURNAL: J. Virol. DATE: 1994 VOLUME: 68 NUMBER: 2 PAGES: 730-7
CODEN: JOVIAM ISSN: 0022-538X LANGUAGE: English

12/3/27 (Item 27 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

118198171 CA: 118(20)198171c PATENT
Genetically engineered cells as universal donor cells for vascular grafts or drug delivery
INVENTOR(AUTHOR): Sims, Peter J.; Bothwell, Alfred L. M.; Elliot, Eileen A.; Flavell, Richard A.; Madri, Joseph; Rollins, Scott; Bell, Leonard; Squinto, Stephen
LOCATION: USA
ASSIGNEE: Oklahoma Medical Research Foundation; Yale University
PATENT: PCT International ; WO 9302188 A1 DATE: 930204
APPLICATION: WO 92US5920 (920714) *US 729926 (910715) *US 906394 (920629)
PAGES: 88 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/00A; C12N-005/12B; A01K-067/027B; C12N-005/16B; C12N-005/22B; C12N-015/87B; A61K-037/00B; C07K-015/00B DESIGNATED COUNTRIES: CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LU; MC; NL; SE

12/3/28 (Item 28 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

116253695 CA: 116(25)253695n JOURNAL
Contribution of the N-linked carbohydrate of erythrocyte antigen CD59 to its complement-inhibitory activity
AUTHOR(S): Ninomiya, Haruhiko; Stewart, Betty H.; Rollins, Scott A.; Zhao, Ji; Bothwell, Alfred L. M.; Sims, Peter J.
LOCATION: Health Sci. Cent., Univ. Oklahoma, Oklahoma City, OK, 73104, USA
JOURNAL: J. Biol. Chem. DATE: 1992 VOLUME: 267 NUMBER: 12 PAGES: 8404-10 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English

12/3/29 (Item 29 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

115277572 CA: 115(25)277572a DISSERTATION
Isolation and characterization of CD59, a membrane attack complex inhibitor of complement
AUTHOR(S): Rollins, Scott Alan
LOCATION: Univ. Oklahoma Health Sci. Cent., Norman, OK, USA
DATE: 1990 PAGES: 192 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int. B 1991, 51(12, Pt. 1), 5802 AVAIL: Univ. Microfilms

Int., Order No. DA9113763

12/3/30 (Item 30 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

115133684 CA: 115(13)133684r JOURNAL
Inhibition of homologous complement by CD59 is mediated by a species-selective recognition conferred through binding to C8 within C5b-8 or C9 within C5b-9
AUTHOR(S): Rollins, Scott A.; Zhao, Ji; Ninomiya, Haruhiko; Sims, Peter J.
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
JOURNAL: J. Immunol. DATE: 1991 VOLUME: 146 NUMBER: 7 PAGES: 2345-51
CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English

12/3/31 (Item 31 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

115129040 CA: 115(13)129040k JOURNAL
Amplified gene expression in CD59-transfected Chinese hamster ovary cells confers protection against the membrane attack complex of human complement
AUTHOR(S): Zhao, Ji; Rollins, Scott A.; Maher, Stephen E.; Bothwell, Alfred L. M.; Sims, Peter J.
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
JOURNAL: J. Biol. Chem. DATE: 1991 VOLUME: 266 NUMBER: 20 PAGES: 13418-22 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English

12/3/32 (Item 32 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

114099628 CA: 114(11)99628t JOURNAL
Regulatory control of the terminal complement proteins at the surface of human endothelial cells: neutralization of a C5b-9 inhibitor by antibody to CD59
AUTHOR(S): Hamilton, Karen K.; Ji, Zhao; Rollins, Scott; Stewart, Betty H.; Sims, Peter J.
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, USA
JOURNAL: Blood DATE: 1990 VOLUME: 76 NUMBER: 12 PAGES: 2572-7
CODEN: BLOOAW ISSN: 0006-4971 LANGUAGE: English

12/3/33 (Item 33 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

113057087 CA: 113(7)57087q JOURNAL
The complement-inhibitory activity of CD59 resides in its capacity to block incorporation of C9 into membrane C5b-9
AUTHOR(S): Rollins, Scott A.; Sims, Peter J.
LOCATION: Health Sci. Cent., Oklahoma Univ., Oklahoma City, OK, 73104, USA
JOURNAL: J. Immunol. DATE: 1990 VOLUME: 144 NUMBER: 9 PAGES: 3478-83
CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English

12/3/34 (Item 34 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)
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111230335 CA: 111(25)230335c JOURNAL
Regulatory control of complement on blood platelets. Modulation of platelet procoagulant responses by a membrane inhibitor of the C5b-9 complex
AUTHOR(S): Sims, Peter J.; Rollins, Scott A.; Wiedmer, Therese
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
JOURNAL: J. Biol. Chem. DATE: 1989 VOLUME: 264 NUMBER: 32 PAGES: 19228-35 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English

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? s s12 and antibod?

             34  S12
        1008678  ANTIBOD?
    S13      16  S12 AND ANTIBOD?
? s s13 and (c5 or c5a)

             16  S13
          19202  C5
           6192  C5A
    S14       9  S13 AND (C5 OR C5A)
? rd s14

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
    S15       9  RD S14 (unique items)
? t s15/7/all
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15/7/1 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 1998 American Chemical Society. All rts. reserv.

127120618 CA: 127(9)120618v CONFERENCE PROCEEDING
Amelioration of lupuslike autoimmune disease in NZB/W F1 mice after treatment with a blocking monoclonal antibody specific for complement component C5
AUTHOR(S): Wang, Yi; Hu, Qile; Madri, Joseph A.; Rollins, Scott A.; Chodera, Amy; Matis, Louis A.
LOCATION: Alexion Pharmaceuticals, 25 Science Park, New Haven, CT, 06511, USA
JOURNAL: Controlling Complement Syst. Novel Drug Dev., (IBC Conf.)
EDITOR: Mazarakis, Helen (Ed), Swart, Sarah Jane (Ed), DATE: 1997
PAGES: 89-109 CODEN: 64QOAM LANGUAGE: English MEETING DATE: 19960000
PUBLISHER: International Business Communications,Southborough, Mass
SECTION:
CA215008 Immunochemistry
IDENTIFIERS: lupus model monoclonal antibody complement C5
DESCRIPTORS:
Monoclonal antibodies...
amelioration of lupus-like autoimmune disease in mice after treatment with blocking monoclonal antibody to complement component C5
Glomerulonephritis...
immune complex; terminal complement cascade role in lupus erythematosus model
Lupus erythematosus...
terminal complement cascade role in lupus erythematosus model
CAS REGISTRY NUMBERS:
80295-53-0 amelioration of lupus-like autoimmune disease in mice after treatment with blocking monoclonal antibody to complement component C5
82986-89-8 terminal complement cascade role in lupus erythematosus model

15/7/2 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

127079964 CA: 127(6)79964q JOURNAL
Inhibition of complement activity by humanized anti-C5 antibody and single-chain Fv
AUTHOR(S): Thomas, Thomas C.; Rollins, Scott A.; Rother, Russell P.; Giannoni, Michelle A.; Hartman, Sandra L.; Elliott, Eileen A.; Nye, Steven H.; Matis, Louis A.; Squinto, Stephen P.; Evans, Mark J.
LOCATION: Alexion Pharmaceuticals, New Haven, CT, 06511, USA
JOURNAL: Mol. Immunol. DATE: 1997 VOLUME: 33 NUMBER: 17/18 PAGES: 1389-1401 CODEN: MOIMD5 ISSN: 0161-5890 PUBLISHER ITEM IDENTIFIER: 0161-5890(96)00078-8 LANGUAGE: English MEETING DATE: 19960000
PUBLISHER: Elsevier
SECTION:
CA215003 Immunochemistry
IDENTIFIERS: complement C5 humanized antibody Fv, single chain Fv antibody complement C5
DESCRIPTORS:
Complement activation...
complement activity inhibition by humanized anti-C5 antibody and single-chain Fv
Humanized antibodies...
monoclonal; complement activity inhibition by humanized anti-C5 antibody and single-chain Fv
Protein sequences...
of anti-complement C5 antibody 5G1.1 heavy and light chain variable regions and single-chain Fv mol. derived from it
DNA sequences...
of anti-complement C5 antibody 5G1.1 heavy and light chain variable regions genes
Antibodies...
single-chain Fv; complement activity inhibition by humanized anti-C5 antibody and single-chain Fv
CAS REGISTRY NUMBERS:
80295-53-0 complement activity inhibition by humanized anti-C5 antibody and single-chain Fv

15/7/3 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125165528 CA: 125(13)165528r JOURNAL
Amelioration of lupus-like autoimmune disease in NZB/W F1 mice after treatment with a blocking monoclonal antibody specific for complement component C5
AUTHOR(S): Wang, Yi; Hu, Qile; Madri, Joseph A.; Rollins, Scott A.; Chodera, Amy; Matis, Louis A.
LOCATION: Immumobiology Program, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1996 VOLUME: 93 NUMBER: 16 PAGES: 8563-8568 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English
SECTION:
CA215008 Immunochemistry
IDENTIFIERS: lupus model monoclonal antibody complement C5
DESCRIPTORS:
Antibodies,monoclonal...
amelioration of lupus-like autoimmune disease in mice after treatment with blocking monoclonal antibody to complement component C5
Kidney,disease, immune complex glomerulonephritis... Lupus erythematosus...

terminal complement cascade role in lupus erythematosus model
CAS REGISTRY NUMBERS:
80295-53-0 amelioration of lupus-like autoimmune disease in mice after treatment with blocking monoclonal antibody to complement component C5
82986-89-8 terminal complement cascade role in lupus erythematosus model

15/7/4 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124143157 CA: 124(11)143157w JOURNAL
Monoclonal antibodies directed against human C5 and C8 block complement-mediated damage of xenogeneic cells and organs
AUTHOR(S): Rollins, Scott A.; Matis, Louis A.; Springhorn, Jeremy P.; Setter, Eva; Wolff, Dennis W.
LOCATION: Department of Immunobiology, Alexion Pharmaceuticals, Inc., New haven, CT, 06511, USA
JOURNAL: Transplantation DATE: 1995 VOLUME: 60 NUMBER: 11 PAGES: 1284-92 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English
SECTION:
CA215004 Immunochemistry
IDENTIFIERS: monoclonal antibody complement C5 C8
DESCRIPTORS:
Antibodies,monoclonal... Blood vessel,disease, endothelium, injury...
Complement... Cytolysis... Heart,disease, injury...
monoclonal antibodies to human C5 and C8 block complement-mediated damage of xenogeneic cells and organs
Transplant and Transplantation,xeno-...
monoclonal antibodies to human C5 and C8 block complement-mediated damage of xenogeneic cells and organs in relation to
CAS REGISTRY NUMBERS:
80295-53-0 80295-58-5 monoclonal antibodies to human C5 and C8 block complement-mediated damage of xenogeneic cells and organs
80295-54-1 role of C5a in complement-mediated damage of xenogeneic cells and organs
82986-89-8 role of C5b-9 in complement-mediated damage of xenogeneic cells and organs

15/7/5 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124143156 CA: 124(11)143156v JOURNAL
Complement inhibition with an anti-C5 monoclonal antibody prevents acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation
AUTHOR(S): Kroshus, Timothy J.; Rollins, Scott A.; Dalmasso, Agustin P.; Elliott, Eileen A.; Matis, Louis A.; Squinto, Stephen P.; Bolman, R. Morton, III
LOCATION: Department of Surgery, University of Minnesota, Minneapolis, MN, USA
JOURNAL: Transplantation DATE: 1995 VOLUME: 60 NUMBER: 11 PAGES: 1194-202 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English
SECTION:
CA215004 Immunochemistry
IDENTIFIERS: cardiac xenotransplant complement monoclonal antibody
DESCRIPTORS:
Antibodies,monoclonal... Complement... Heart,xenotransplant... Swine...
Transplant and Transplantation,xeno-...
complement inhibition with an anti-C5 monoclonal antibody prevents acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation
CAS REGISTRY NUMBERS:

80295-54-1 complement inhibition with an anti-C5 monoclonal antibody prevents acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation
82986-89-8 role of complement C5b-9 in acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation

15/7/6 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124127101 CA: 124(10)127101t PATENT
Anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
INVENTOR(AUTHOR): Evans, Mark J.; Matis, Louis; Mueller, Eileen Elliott; Nye, Steven H.; Rollins, Scott; Rother, Russell P.; Springhorn, Jeremy P.; Squinto, Stephen P.; Thomas, Thomas C.; et al.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9529697 A1 DATE: 951109
APPLICATION: WO 95US5688 (950501) *US 236208 (940502)
PAGES: 159 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-038/36A; A61K-039/00B; A61K-039/395B; C07K-014/00B; C07K-014/75B; C07K-016/00B; C07K-016/18B; C07K-016/36B; C07K-016/46B; C12N-005/10B; C12N-005/20B; C12N-015/09B; C12N-015/10B; C12N-015/13B; C12N-015/63B; C12P-021/02B; C12P-021/08B DESIGNATED COUNTRIES: AM; AU; BB; BG; BR; BY; CA; CN; CZ; EE; FI; GE; HU; IS; JP; KG; KP; KR; KZ; LK; LR; LT; LV; MD; MG; MN; MX; NO; NZ; PL; RO; RU; SG; SI; SK; TJ; TM; TT; UA; UG; US; UZ; VN
DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG
SECTION:
CA263003 Pharmaceuticals
CA203XXX Biochemical Genetics
CA215XXX Immunochemistry
IDENTIFIERS: antibody complement C5 cloning glomerulonephritis sequence
DESCRIPTORS:
Antibodies,monoclonal... Deoxyribonucleic acid sequences,complementary... Hybridoma... Immunoglobulins,G... Kidney,disease, glomerulonephritis... Molecular cloning... Packaging materials... Polymerase chain reaction... Protein sequences...
anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
Immune complexes...
deposition of; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
Proteins,metabolic disorders, proteinuria,biological studies...
inhibition of; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
Antigens...
KSSKC epitope, antibodies binding to; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
CAS REGISTRY NUMBERS:
172893-24-2P 173011-96-6P 173012-07-2 173012-10-7P 173012-12-9P 173012-14-1P 173012-17-4P 173012-19-6P 173012-21-0P 173012-23-2P 173012-25-4P 173012-27-6P 173012-29-8P amino acid sequence; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
80295-53-0 antibodies to; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
172998-82-2P epitope KSSKC-contg. antigen; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
173012-09-4P 173012-11-8P 173012-13-0P 173012-15-2P 173012-16-3P 173012-18-5P 173012-20-9P 173012-22-1P 173012-24-3P 173012-26-5P 173012-28-7P 173012-30-1P 173146-43-5 173146-44-6 173146-45-7

nucleic acid sequence; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
173016-57-4 PCR primer UDEC395; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
173016-56-3 PCR primer UDEC690; anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases

15/7/7 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123337462 CA: 123(25)337462s PATENT
Method for reducing immune and hemostatic dysfunctions during extracorporeal circulation
INVENTOR(AUTHOR): Rollins, Scott A.; Smith, Brian R.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.; Yale University
PATENT: PCT International ; WO 9525540 A1 DATE: 950928
APPLICATION: WO 95US3614 (950322) *US 217391 (940323)
PAGES: 34 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/00A; A61K-039/395B; C07K-016/00B; C07K-016/18B DESIGNATED COUNTRIES: AU; CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
SECTION:
CA215003 Immunochemistry
IDENTIFIERS: monoclonal antibody complement C5 extracorporeal circulation
DESCRIPTORS:
Antibodies,monoclonal... Circulation,extracorporeal...
Circulation,extracorporeal, cardiopulmonary bypass...
monoclonal anti-C5 antibody for reducing immune and hemostatic dysfunctions during extracorporeal circulation
CAS REGISTRY NUMBERS:
80295-43-8 80295-53-0 80295-54-1 80295-55-2 monoclonal anti-C5 antibody for reducing immune and hemostatic dysfunctions during extracorporeal circulation

15/7/8 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123196481 CA: 123(15)196481h JOURNAL
Anti-C5 monoclonal antibody therapy prevents collagen-induced arthritis and ameliorates established disease
AUTHOR(S): Wang, Yi; Rollins, Scott A.; Madri, Joseph A.; Matis, Louis A.
LOCATION: Immunobiol. Program, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1995 VOLUME: 92
NUMBER: 19 PAGES: 8955-9 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English
SECTION:
CA215008 Immunochemistry
IDENTIFIERS: arthritis C5 complement monoclonal antibody
DESCRIPTORS:
Antibodies,monoclonal... Arthritis... Arthritis,rheumatoid...
Collagens,type II,biological studies...
anti-C5 complement monoclonal antibody therapy prevents collagen-induced arthritis and ameliorates established disease
CAS REGISTRY NUMBERS:
80295-53-0 anti-C5 complement monoclonal antibody therapy prevents collagen-induced arthritis and ameliorates established disease

15/7/9 (Item 9 from file: 399)

123141260 CA: 123(11)141260e JOURNAL

Rapid expression of an anti-human C5 chimeric Fab utilizing a vector that replicates in COS and 293 cells

AUTHOR(S): Evans, Mark J.; Hartman, Sandra L.; Wolff, Dennis W.; Rollins, Scott A.; Squinto, Stephen P.

LOCATION: Department of Molecular Development, Alexion Pharmaceuticals, Inc., 25 Science Park, New Haven, USA

JOURNAL: J. Immunol. Methods DATE: 1995 VOLUME: 184 NUMBER: 1 PAGES: 123-38 CODEN: JIMMBG ISSN: 0022-1759 LANGUAGE: English

SECTION:
CA215003 Immunochemistry

IDENTIFIERS: pAPEX3P vector antibody Fab C5 complement

DESCRIPTORS:

Antibodies,monoclonal...
Fab; rapid expression of anti-human C5 chimeric Fab by pAPEX-3P vector in COS and 293 cells and ex vivo model of complement-mediated tissue damage

Genetic vectors...
pAPEX-3P; rapid expression of anti-human C5 chimeric Fab by pAPEX-3P vector in COS and 293 cells and ex vivo model of complement-mediated tissue damage

Animal cell line,COS... Animal cell line,293...
rapid expression of anti-human C5 chimeric Fab by pAPEX-3P vector in COS and 293 cells and ex vivo model of complement-mediated tissue damage

Injury...
tissue; rapid expression of anti-human C5 chimeric Fab by pAPEX-3P vector in COS and 293 cells and ex vivo model of complement-mediated tissue damage

CAS REGISTRY NUMBERS:

80295-53-0 rapid expression of anti-human C5 chimeric Fab by pAPEX-3P vector in COS and 293 cells and ex vivo model of complement-mediated tissue damage

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?begin 55,72,154,399,351

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            $0.06    0.002 Hrs File1
     $0.06  Estimated cost File1
     $0.06  Estimated cost this search
     $0.06  Estimated total session cost   0.002 Hrs.

SYSTEM:OS  - DIALOG OneSearch
  File  55:BIOSIS PREVIEWS(R)  1985-1996/Nov W4
        (c) 1996 BIOSIS
  File  72:EMBASE  1985-1996/Iss 48
        (c) 1996 Elsevier Science B.V.
  File 154:MEDLINE(R)  1985-1996/Dec W4
        (c) format only 1996 Knight-Ridder Info
*File 154: MEDLINE updates delayed. See HELP DELAY 154
  File 399:CA SEARCH(R)  1967-1996/UD=12524
        (c) 1996 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
For format prices, including formats 6 & 8, see HELP RATES 399.
  File 351:DERWENT WPI  1981-1996/UD=9648;UA=9645;UM=9637
        (c)1996 Derwent Info Ltd

      Set  Items  Description
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         112006  COMPLEMENT
              0  5GH6K
              0  5G27K
              1  KSSKC
      S1      1  C5 AND COMPLEMENT AND (5GH6K OR 5G27K OR KSSKC)
?t s1/3/all


 1/3/1     (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 1996 American Chemical Society. All rts. reserv.

  124127101    CA: 124(10)127101t    PATENT
  Anti-complement C5 antibodies for the treatment of glomerulonephritis and
other inflammatory diseases
  INVENTOR(AUTHOR): Evans, Mark J.; Matis, Louis; Mueller, Eileen Elliott;
Nye, Steven H.; Rollins, Scott; Rother, Russell P.; Springhorn, Jeremy P.;
Squinto, Stephen P.; Thomas, Thomas C.; et al.
  LOCATION: USA
  ASSIGNEE: Alexion Pharmaceuticals, Inc.
  PATENT: PCT International ; WO 9529697 A1  DATE: 951109
  APPLICATION: WO 95US5688 (950501) *US 236208 (940502)
  PAGES: 159 pp.  CODEN: PIXXD2  LANGUAGE: English  CLASS: A61K-038/36A;
A61K-039/00B; A61K-039/395B; C07K-014/00B; C07K-014/75B; C07K-016/00B;
C07K-016/18B; C07K-016/36B; C07K-016/46B; C12N-005/10B; C12N-005/20B;
C12N-015/09B; C12N-015/10B; C12N-015/13B; C12N-015/63B; C12P-021/02B;
C12P-021/08B  DESIGNATED COUNTRIES: AM; AU; BB; BG; BR; BY; CA; CN; CZ; EE;
FI; GE; HU; IS; JP; KG; KP; KR; KZ; LK; LR; LT; LV; MD; MG; MN; MX; NO; NZ;
PL; RO; RU; SG; SI; SK; TJ; TM; TT; UA; UG; US; UZ; VN
  DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB;
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GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG
?s c5(10n)(hydrolysis or tryptic)(10n)(alpha(w)chain) and complement

```
          15959  C5
         266161  HYDROLYSIS
          18365  TRYPTIC
        1020867  ALPHA
         556598  CHAIN
              3  C5(10N)(HYDROLYSIS OR TRYPTIC)(10N)ALPHA(W)CHAIN
         112006  COMPLEMENT
      S2       3  C5(10N)(HYDROLYSIS OR TRYPTIC)(10N)(ALPHA(W)CHAIN) AND
                  COMPLEMENT
?rd s2

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
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 3/3/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
(c) 1996 BIOSIS. All rts. reserv.

6510438     BIOSIS Number: 85110959
  MOLECULAR ANALYSIS OF HUMAN COMPLEMENT COMPONENT C5 LOCALIZATION OF THE
STRUCTURAL GENE TO CHROMOSOME 9
  WETSEL R A; LEMONS R S; LE BEAU M M; BARNUM S R; NOACK D; TACK B F
  DEP. PEDIATR., WASH. UNIV. SCH. MED., ST. LOUIS, MO. 63110.
  BIOCHEMISTRY 27 (5). 1988.  1474-1482.   CODEN: BICHA
  Full Journal Title:  Biochemistry
  Language: ENGLISH
?t s3/7/1


 3/7/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
(c) 1996 BIOSIS. All rts. reserv.

6510438     BIOSIS Number: 85110959
  MOLECULAR ANALYSIS OF HUMAN COMPLEMENT COMPONENT C5 LOCALIZATION OF THE
STRUCTURAL GENE TO CHROMOSOME 9
  WETSEL R A; LEMONS R S; LE BEAU M M; BARNUM S R; NOACK D; TACK B F
  DEP. PEDIATR., WASH. UNIV. SCH. MED., ST. LOUIS, MO. 63110.
  BIOCHEMISTRY 27 (5). 1988.  1474-1482.   CODEN: BICHA
  Full Journal Title:  Biochemistry
  Language: ENGLISH
  A human C5 clone (pC5HG2) was isolated from a cDNA library constructed
from Hep G2 mRNA. The DNA sequence showed that the pC5HG2 insert was
comprised of 3309 base pairs of pro-C5 coding sequence and 404 base pairs
of 3'-untranslated sequence. The derived amino acid sequence contained the
entire coding sequence of the C5 .alpha.-chain, the .beta.-.alpha.-chain
junction region, and 100 amino acids (approximately 50%) of the
.beta.-chain. Protein sequences of four C5 tryptic peptides were aligned
exactly to this sequence and demonstrated that C5 synthesized and secreted
```

by Hep G2 cells is probably identical with plasma-derived C5. Coding sequence alignment of the human C5 sequences with those of murine C5 indicated that 80% of the nucleotides and 79% of the amino acids were placed identically in the two species. Amino acid sequence alignment of the homologous family members C3, C4, and .alpha.2-macroglobulin with that of C5 demonstrated 27%, 25%, and 19% identity, respectively. As was found in murine C5, the corresponding thiol ester region of human C5 contained several conserved amino acids, but the critical cysteine and glutamine residues which give rise to the intramolecular thiol ester bond in C3, C4, and .alpha.2-macroglobulin were absent in C5, having been replaced by serine and alanine, respectively. With the use of a panel of hamster-human somatic cell hybrids, the C5 gene was mapped to human chromosome 9. In situ chromosomal hybridization studies employing metaphase cells further localized the gene to bands 9q32-34, with the largest cluster of grains at 9q34.1.

?s c5 and alpha and (tryptic or hydrolysis) and complement

```
          15959  C5
        1020867  ALPHA
          18365  TRYPTIC
         266161  HYDROLYSIS
         112006  COMPLEMENT
      S4      10  C5 AND ALPHA AND (TRYPTIC OR HYDROLYSIS) AND COMPLEMENT
?rd s4

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S5       4  RD S4 (unique items)
?t s5/7/all


 5/7/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
(c) 1996 BIOSIS. All rts. reserv.

11572126     BIOSIS Number: 98172126
  Isolation, primary structure, and evolution of the third component of
chicken complement and evidence for a new member of the
alpha-2-macroglobulin family
  Mavroidis M; Sunyer J O; Lambris J D
  Lab. Protein Chem., Dep. Pathol. Lab. Med., Univ. Pennsylvania,
Philadelphia, PA 19104-6079, USA
  Journal of Immunology 154 (5). 1995.  2164-2174.
  Full Journal Title:  Journal of Immunology
  ISSN: 0022-1767
  Language: ENGLISH
  Print Number: Biological Abstracts Vol. 099 Iss. 008 Ref. 112429
```

Although the third component of complement, C3, has been isolated and its primary structure determined from most living classes of vertebrate, limited information is available on its structure and function for aves, which represent a significant stage in complement evolution. In this study, we present the complete cDNA sequence of chicken C3, the cDNA sequences of the thioester region for two chicken alpha-2-macroglobulin (alpha-2M)-related proteins, a simplified method for purifying chicken C3, and an analysis of the C3 convertase and factor 1-mediated cleavages in chicken C3. Using the reverse-transcriptase PCR, with degenerate

oligonucleotide primers derived from two conserved C3 sequences (GCGEQN/TM, TWLTAY/FV) and liver mRNA as template, we isolated three distinct 220-bp PCR products, one with a high degree of sequence similarity to C3 and two to alpha-2M and pregnancy zone protein from other species. The complete cDNA sequence of chicken C3 was obtained by screening a chicken liver lambda-gt10 library with the C3 PCR product and probes from the 5' end of the partial-length C3 clones. The obtained sequence is in complete agreement with the protein sequence of several tryptic peptides of purified chicken C3. Chicken pro-C3 consists of an 18-residue putative signal peptide, a 640-residue beta-chain (70 kDa), a 989-residue alpha-chain (111 kDa), and an RKRR linker region. It contains an internal thioester and three potential N-glycosylation sites, all in the a-chain. The convertase cleavage site, predicted to be Arg-Ser, was confirmed by sequencing the zymosan-bound C3 fragments generated upon complement activation. NH-2-terminal sequencing of the purified C3 chains showed that 1) pro-C3 is indeed cleaved at the RKRR linker sequence to generate the mature two-chain molecule, and 2) the beta-chain of chicken C3 is blocked. The deduced amino acid sequence shows 54, 54, 54, 53, 52, 57, and 55% amino acid identities to human, mouse, rat, guinea pig, rabbit, cobra, and Xenopus C3, respectively, and an identity of 44, 31, and 33% to trout, hagfish, and lamprey C3, respectively. The identities to human C4, C5, and alpha-2M are 31, 29 and 23%, respectively. A phylogenetic tree for C3, C4, C5, and alpha-2M-related proteins was constructed based on the sequence data and is discussed.

5/7/2 (Item 2 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

10874870 BIOSIS Number: 97074870
Third component of trout complement: cDNA cloning and conservation of functional sites
Lambris J D; Lao Z; Pang J; Alsenz J
Dep. Pathol. and Lab. Med., Univ. Pa., 410 Johnson Pavillion, 36th and Hamilton Walk, Philadelphia, PA 19104, USA
Journal of Immunology 151 (11). 1993. 6123-6134.
Full Journal Title: Journal of Immunology
ISSN: 0022-1767
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 004 Ref. 042364
Of the 30 distinct complement proteins recognized to date, C3 is probably the most versatile and multifunctional molecule known, interacting with at least 20 different proteins. It plays a critical role in both pathways of complement activation and participates in phagocytic and immunoregulatory processes. Structural and functional analysis of C3 from different species, in addition to phylogenetic information, provides insights into the structural elements mediating the various functions. This study describes the cDNA cloning of one of two isoforms of the third complement component, C3-1, of rainbow trout (Salmo gairdneri) and the analysis of its functional sites. By screening a trout liver lambda-gt11 library with anti-trout C3 chain-specific antibodies and polymerase chain reaction we have determined the cDNA sequence of trout C3-1. The obtained sequence is in complete agreement with the protein sequence of several tryptic peptides, corresponding to different regions of trout C3-1. C3-1 consists of 1640 amino acids with a calculated molecular mass of 181,497 Da. The sequence contains two potential N-glycocylation sites, one on each chain of C3. The deduced protein sequence showed 44.1, 43.3, 44.2, 44.9, 43.1, 43.8, 45.9,

29.9, and 33.1 % amino acid identities to human, mouse rat, guinea pig, rabbit, cobra, frog, hagfish, and lamprey C3, whereas the identities to human C4, C5, and alpha-2M are 30.4, 28, and 22.9%, respectively. The trout C3 amino acid sequence shows clusters of high and low similarity to C3 from other species. In the regions of high similarity belong the C3 domains that contain the thiolester site and the properdin binding sites, whereas the regions that correspond to regions of human C3 where CR1 and CR2 bind show low amino acid sequence similarity. The deduced amino acid sequence shows that the C3 convertase cleavage site (Arg-Ser) is conserved in trout C3, whereas the factor I cleavage sites are Arg-Ala and Arg-Thr instead of Arg-Ser, which is found in the C3 of other species. Protein sequencing of the trout C3 fragments fixed on zymosan during complement activation confirmed the cleavage of trout C3 by trout C3 convertase and factor I at Arg-Ser and Arg-Thr, respectively.

5/7/3 (Item 3 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

6510438 BIOSIS Number: 85110959
MOLECULAR ANALYSIS OF HUMAN COMPLEMENT COMPONENT C5 LOCALIZATION OF THE STRUCTURAL GENE TO CHROMOSOME 9
WETSEL R A; LEMONS R S; LE BEAU M M; BARNUM S R; NOACK D; TACK B F
DEP. PEDIATR., WASH. UNIV. SCH. MED., ST. LOUIS, MO. 63110.
BIOCHEMISTRY 27 (5). 1988. 1474-1482. CODEN: BICHA
Full Journal Title: Biochemistry
Language: ENGLISH
A human C5 clone (pC5HG2) was isolated from a cDNA library constructed from Hep G2 mRNA. The DNA sequence showed that the pC5HG2 insert was comprised of 3309 base pairs of pro-C5 coding sequence and 404 base pairs of 3'-untranslated sequence. The derived amino acid sequence contained the entire coding sequence of the C5 .alpha.-chain, the .beta.-.alpha.-chain junction region, and 100 amino acids (approximately 50%) of the .beta.-chain. Protein sequences of four C5 tryptic peptides were aligned exactly to this sequence and demonstrated that C5 synthesized and secreted by Hep G2 cells is probably identical with plasma-derived C5. Coding sequence alignment of the human C5 sequences with those of murine C5 indicated that 80% of the nucleotides and 79% of the amino acids were placed identically in the two species. Amino acid sequence alignment of the homologous family members C3, C4, and .alpha.2-macroglobulin with that of C5 demonstrated 27%, 25%, and 19% identity, respectively. As was found in murine C5, the corresponding thiol ester region of human C5 contained several conserved amino acids, but the critical cysteine and glutamine residues which give rise to the intramolecular thiol ester bond in C3, C4, and .alpha.2-macroglobulin were absent in C5, having been replaced by serine and alanine, respectively. With the use of a panel of hamster-human somatic cell hybrids, the C5 gene was mapped to human chromosome 9. In situ chromosomal hybridization studies employing metaphase cells further localized the gene to bands 9q32-34, with the largest cluster of grains at 9q34.1.

5/7/4 (Item 4 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

6415858 BIOSIS Number: 85016379

THE CHEMICAL STRUCTURE OF THE C4D FRAGMENT OF THE HUMAN COMPLEMENT COMPONENT C4
CHAKRAVARTI D N; CAMPBELL R D; PORTER R R
DEP. IMMUNOL., RES. INST. SCRIPPS CLINIC, 10666 N. TORREY PINES RD., LA JOLLA, CALIF. 92037, USA.
MOL IMMUNOL 24 (11). 1987. 1187-1198. CODEN: MOIMD
Full Journal Title: Molecular Immunology
Language: ENGLISH
The complete amino acid sequence of the C4d fragment (380 residues long) of the human complement component C4 is presented. Most of the sequence was determined by analysis of CNBr peptides and tryptic peptides obtained from S-carboxymethylated protein. The sequence of the amino terminal 88 residues [Campbell R. D., Gagnon J. and Porter R. R. (1981) Biochem. J. 199, 359-370] and a 106 residue polymorphic segment of C4d [Chakravarti D.N., Campbell R.D. and Gagnon J. (1983) FEBS Lett. 154, 387-390] was extended. Some overlaps not provided by the protein sequence analysis were obtained from the amino acid sequence predicted by the nucleotide sequence [Belt K. T., Carroll M.C. and Porter R.R. (1984) Cell 36, 907-914]. The present protein sequence data provide information for the isolation of all the CNBr and succinylated tryptic peptides of C4d. In addition to the polymorphism previously described, two other sets of polymorphic amino acid residues at positions 153 (Ile/Ser) and 154 (Gln/Ala) have been identified. The major site of glycosylation has been shown to be an asparagine residue located in the sequence - Asn- Val- Thr- in the carboxy terminal end of C4d. A remarkable difference in the predicted secondary structure of C4d arising from one set of four polymorphic residues in a stretch of six residues and another single polymorphic residue suggests a structural basis for the origin of the different chemical reactivies of the C4 isotypes (C4A and C4B) and their serological difference in the expression of Rodgers or Chido blood group antigens. Possible non-covalent membrane attachment sites have been suggested from the hydropathy profile. Comparison of the C4d sequence with human C3, C5 and .alpha.2-macroglobulin revealed extended stretches of sequence similarity (between 19 and 38% homology) with the corresponding regions of these proteins.
?s s5 and (27 or 46) and complement

```
                4  S5
           371396  27
           206639  46
           112006  COMPLEMENT
      S6        1  S5 AND (27 OR 46) AND COMPLEMENT
?t s6/3/all


 6/3/1     (Item 1 from file: 55)
DIALOG(R)File  55:BIOSIS PREVIEWS(R)
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6510438     BIOSIS Number: 85110959
  MOLECULAR ANALYSIS OF HUMAN COMPLEMENT COMPONENT C5 LOCALIZATION OF THE STRUCTURAL GENE TO CHROMOSOME 9
  WETSEL R A; LEMONS R S; LE BEAU M M; BARNUM S R; NOACK D; TACK B F
  DEP. PEDIATR., WASH. UNIV. SCH. MED., ST. LOUIS, MO. 63110.
  BIOCHEMISTRY 27 (5). 1988. 1474-1482.   CODEN: BICHA
  Full Journal Title: Biochemistry
  Language: ENGLISH
?s complement and antibod? and 5g1(w)1
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          914112  ANTIBOD?
              13  5G1
         7472928  1
               1  5G1(W)1
      S7       1  COMPLEMENT AND ANTIBOD? AND 5G1(W)1
?t s7/3/all


 7/3/1     (Item 1 from file: 351)
DIALOG(R)File 351:DERWENT WPI
(c)1996 Derwent Info Ltd. All rts. reserv.

010491522  WPI Acc No: 95-392923/50
XRAM Acc No: C95-169278
    Treating glomerulonephritis with antibody against complement C5
    component  - to inhibit complement induced cell lysis
Patent Assignee: (ALEX-) ALEXION PHARM INC
Author (Inventor): EVANS M J; MATIS L; MUELLER E E; NYE S H; ROLLINS S;
    ROTHER R P; SPRINGHORN J P; SQUINTO S P; THOMAS T C; WANG Y; WILKINS J
    A
Patent Family:
    CC Number     Kind     Date      Week
    WO 9529697      A1     951109     9550   (Basic)
    AU 9524747      A      951129     9609

Priority Data (CC No Date): US 236208 (940502)
Applications (CC,No,Date): WO 95US5688 (950501); AU 9524747 (950501)
?s c5 and complement and antibod?

          15959  C5
         112006  COMPLEMENT
         914112  ANTIBOD?
      S8    1102  C5 AND COMPLEMENT AND ANTIBOD?
?s c5(10n)antibod? and complement

          15959  C5
         914112  ANTIBOD?
            606  C5(10N)ANTIBOD?
         112006  COMPLEMENT
      S9     375  C5(10N)ANTIBOD? AND COMPLEMENT
?s s9 and alpha(w)chain

            375  S9
        1020867  ALPHA
         556598  CHAIN
          14545  ALPHA(W)CHAIN
     S10      9  S9 AND ALPHA(W)CHAIN
?rd s9

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
...examined 50 records  (50)


INT CONF
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INT CONF

!

INT CONF
!
?rd s10

>>>Duplicate detection is not supported for File 351.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
     S11   4 RD S10 (unique items)
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 11/3/1   (Item 1 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)
(c) 1996 BIOSIS. All rts. reserv.

10076142   BIOSIS Number: 95076142
  MOLECULAR BASIS OF COMPLEMENT RESISTANCE OF HUMAN MELANOMA CELLS
EXPRESSING THE C3-CLEAVING MEMBRANE PROTEASE P65
  OLLERT M W; KADLEC J V; PETRELLA E C; BREDEHORST R; VOGEL C-W
  DEP. BIOCHEM. MOLECULAR BIOL., UNIV. HAMBURG, MARTIN-LUTHER-KING-PL. 6,
2000 HAMBURG 13, GER.
  CANCER RES 53 (3). 1993. 592-599.   CODEN: CNREA
  Full Journal Title: Cancer Research
  Language: ENGLISH

 11/3/2   (Item 2 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)
(c) 1996 BIOSIS. All rts. reserv.

9603458   BIOSIS Number: 94108458
  FORMATION AND STRUCTURE OF THE C5B-7 COMPLEX OF THE LYTIC PATHWAY OF
COMPLEMENT
  DISCIPIO R G
  DEP. IMMUNOLOGY IMM18, RESEARCH INSTITUTE SCRIPPS CLINIC, 10666 N. TORREY
PINES RD., LA JOLLA, CALIF. 92037.
  J BIOL CHEM 267 (24). 1992. 17087-17094.   CODEN: JBCHA
  Full Journal Title: Journal of Biological Chemistry
  Language: ENGLISH

 11/3/3   (Item 3 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)
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5913963   BIOSIS Number: 84046528
  COVALENT ASSOCIATION OF C3B WITH C4B WITHIN C5 CONVERTASE OF THE
CLASSICAL COMPLEMENT PATHWAY
  TAKATA Y; KINOSHITA T; KOZONO H; TAKEDA J; TANAKA E; HONG K; INOUE K
  DEP. BACTERIOLOGY, OSAKA UNIV. MED. SCH., SUITA, OSAKA 565, JAPAN.
  J EXP MED 165 (6). 1987. 1494-1507.   CODEN: JEMEA
  Full Journal Title: Journal of Experimental Medicine

Language: ENGLISH

11/3/4 (Item 1 from file: 351)
DIALOG(R)File 351:DERWENT WPI

010491522 WPI Acc No: 95-392923/50
XRAM Acc No: C95-169278
Treating glomerulonephritis with antibody against complement C5 component - to inhibit complement induced cell lysis
Patent Assignee: (ALEX-) ALEXION PHARM INC
Author (Inventor): EVANS M J; MATIS L; MUELLER E E; NYE S H; ROLLINS S; ROTHER R P; SPRINGHORN J P; SQUINTO S P; THOMAS T C; WANG Y; WILKINS J A
Patent Family:

CC Number	Kind	Date	Week	
WO 9529697	A1	951109	9550	(Basic)
AU 9524747	A	951129	9609	

Priority Data (CC No Date): US 236208 (940502)
Applications (CC,No,Date): WO 95US5688 (950501); AU 9524747 (950501)
?t s11/7/1-3

11/7/1 (Item 1 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

10076142 BIOSIS Number: 95076142
MOLECULAR BASIS OF COMPLEMENT RESISTANCE OF HUMAN MELANOMA CELLS EXPRESSING THE C3-CLEAVING MEMBRANE PROTEASE P65
OLLERT M W; KADLEC J V; PETRELLA E C; BREDEHORST R; VOGEL C-W
DEP. BIOCHEM. MOLECULAR BIOL., UNIV. HAMBURG, MARTIN-LUTHER-KING-PL. 6, 2000 HAMBURG 13, GER.
CANCER RES 53 (3). 1993. 592-599. CODEN: CNREA
Full Journal Title: Cancer Research
Language: ENGLISH
The molecular mechanism of complement resistance of the human SK-MEL-170 melanoma cell line was investigated. The cells have been shown to express the C3b-cleaving membrane protease p65. To delineate the molecular consequences of the C3b-cleaving activity for the complement cytotoxicity, the molecular events during the initiation (R24 monoclonal antibody, C1), amplification (C4, C3), and membrane attack (C5, C9) phases of complement were studied in comparison to a complement-susceptible human melanoma line (SK-MEL-93-2). No cleavage of C4b and C5b, 2 molecules structurally similar to C3b, was observed on the cells during classical pathway activation indicating the specificity of the p65 protease for the C3b molecule. The rapid degradation of C3b by p65 on the surface of complement-resistant SK-MEL-170 cells generates a Mr 30,000 C3.alpha.'-chain-fragment detectable as early as 1 min after complement activation, whereas no such fragment was present in detectable amounts on complement-susceptible cells. As a result of the rapid C3b proteolysis by p65 on resistant SK-MEL-170 cells, less C5 convertases are formed, which in turn results in the formation of a lower number of terminal complement components and membrane attack complexes. R24 antibody and C1q binding to the resistant cells was slightly lower as to susceptible cells. C4 binding studies, however, revealed that the observed difference in antibody and C1q binding has no influence on the complement

resistance of SK-MEL-170 cells: significantly more C4b was bound to complement-resistant (1565 .+-. 92 fg/cell) as compared to susceptible cells (715 .+-. 31 fg/cell). On extraction of the molecular forms of C4 bound to the cell membranes, an additional high molecular weight C4 species.sbd.apparently a C4b-C4b homodimer.sbd.appeared only on the resistant SK-MEL-170 cells that may function as a residual back-up C5 convertase. Collectively, these results show that SK-MEL-170 human melanoma cells evade complement-mediated cytolysis despite sufficient activation of early components of the classical complement pathway by p65-mediated rapid degradation of surface-bound C3b, leading to a significant reduction in membrane attack complex formation. Thus, rapid cleavage of surface deposited C3b was established as a powerful mechanism of complement resistance.

11/7/2 (Item 2 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

9603458 BIOSIS Number: 94108458
FORMATION AND STRUCTURE OF THE C5B-7 COMPLEX OF THE LYTIC PATHWAY OF COMPLEMENT
DISCIPIO R G
DEP. IMMUNOLOGY IMM18, RESEARCH INSTITUTE SCRIPPS CLINIC, 10666 N. TORREY PINES RD., LA JOLLA, CALIF. 92037.
J BIOL CHEM 267 (24). 1992. 17087-17094. CODEN: JBCHA
Full Journal Title: Journal of Biological Chemistry
Language: ENGLISH
The formation and structure of the complement cytolytic intermediary complex, C5b-7, were studied with the aim of determining the interactive regions of C5, C6, and C7. The structure of human complement component C5 was elucidated by the application of limited proteolysis which generated well characterized major polypeptide fragments of this molecule. Plasmin, thrombin, and kallikrein cleave C5b with greater facility than C5. The most useful cleavage of C5b was effected by plasmin because the fragmentation pattern was similar to the processing the C3b by factors H, I, and kallikrein. Plasmin hydrolyzes peptide bonds within the .alpha.'-chain of C5b, resulting in a four-chain fragment, C5c (Mr = 142,000), and a single chain fragment, C5d (Mr = 43,000). Circular dichroism spectroscopic analyses indicated that C5d is substantially richer in .alpha.-helical content than is C5c (27 versus 9%). Polyclonal antibodies directed against C5c blocked the interaction of C5b-6 with C7, whereas antibodies directed against C5d inhibited the binding of C5 with C3b. Chemical cross-linking using a cleavable radioiodinated photoreactive reagent revealed that both C6 and C7 associate preferentially with the .alpha.'-chain of C5b. The reversible interactions of C5 with C6, C7, and major polypeptide fragments derived from these were investigated with solid phase binding assays. The results indicate that the carboxyl-terminal domains of C6 and C7, which have cysteine-rich modules homologous to those found in factors H and I, have the capacity to link specifically with C5.

11/7/3 (Item 3 from file: 55)
DIALOG(R)File 55:BIOSIS PREVIEWS(R)

5913963 BIOSIS Number: 84046528
COVALENT ASSOCIATION OF C3B WITH C4B WITHIN C5 CONVERTASE OF THE

CLASSICAL COMPLEMENT PATHWAY

TAKATA Y; KINOSHITA T; KOZONO H; TAKEDA J; TANAKA E; HONG K; INOUE K
DEP. BACTERIOLOGY, OSAKA UNIV. MED. SCH., SUITA, OSAKA 565, JAPAN.

J EXP MED 165 (6). 1987. 1494-1507. CODEN: JEMEA
Full Journal Title: Journal of Experimental Medicine
Language: ENGLISH

The C convertase of the classical complement pathway is a complex enzyme consisting of three complement fragments, C4b, C2a, and C3b. Previous studies have elucidated functional roles of each subunit (4, 6, 7), but, little is known about how the subunits associate with each other. In this investigation, we studied the nature of the classical C% convertase that was assembled on sheep erythrocytes. We found that one of the nascent C3b molecule that had been generated by the C3 convertase directly bound covalently to C4b. C3b bound to the .alpha.' chain of C4b through an ester bond, which could be cleaved by treatment with hydroxylamine. The ester bond was rather unstable, with a half-life of 7.9 h at pH 7.4 and 37% C. Formation of the C4b-C3b dimer is quiet efficient; e.g., 54% of the cell-bound C3b was associated with C4b when 25,000 molecules of C4b and 12,000 molecules of C3b were present per cell. Kinetic analysis also showed the efficient formation of the C4b-C3b dimer; the rate of dimer formation was similar to or even faster than that of cell-bound monomeric C3b molecules. These results indicate that the C4b is a highly reactive acceptor molecule for nascent C3b. High-affinity C5-binding site with an association constant of 2.1 .times. 108 L/M were demonstrated on C4b-C3b dimer-bearing sheep erthocytes, EAC43 cells. The number of high-affinity C5-binding sites coincided with the number of C4b-C3b dimers, but not with the total number of cell-bound C3b molecules. Anti-C4 antibodies caused 80% inhibition of the binding of C5 to EAC43 cells. These results suggest that only C4b-associated C3b serves as a high-affinity C5 binding site. EAC14 cells had a small amount of high-affinity C5 binding sites with an association constant of 8.1 .times. 107 L/M 100 molecules of bound C4b being necessary for 1 binding site. In accordance with the hypothesis that C4b-associated C4b might also serve as a high-affinity C5-binding site, a small amount of C4b-C4b dimer was detected on EAC14 cells by SDS-PAE analysis. Taken together, these observations indicate that high-affinity binding of C5 is probably divalent, in that C5 recognizes both promoters with dimers. The high-affinity binding may allow selective binding of C5 to the convertase in spite of surrounding monomeric C3b molecules.

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?e au=rollins, scott ?

Ref	Items	Index-term
E1	5	AU=ROLLINS, SCOTT
E2	0	*AU=ROLLINS, SCOTT ?
E3	32	AU=ROLLINS, SCOTT A.
E4	1	AU=ROLLINS, SCOTT ALAN
E5	3	AU=ROLLINS, SEAN M.
E6	1	AU=ROLLINS, STEPHEN
E7	1	AU=ROLLINS, T. E.
E8	1	AU=ROLLINS, THOMAS
E9	12	AU=ROLLINS, THOMAS E.
E10	1	AU=ROLLINS, TREVEN
E11	10	AU=ROLLINS, V.
E12	1	AU=ROLLINS, VICTOR

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?s s1-s4

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S12	11	S1-S4

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	5	AU=ROLLINS, SCOTT
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	32	AU=ROLLINS, SCOTT A.
	1	AU=ROLLINS, SCOTT ALAN
S13	38	E1-E4

?rd s13

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S14	37	RD S13 (unique items)

?t s14/3/all

14/3/1 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125165528 CA: 125(13)165528r JOURNAL

Amelioration of lupus-like autoimmune disease in NZB/W F1 mice after treatment with a blocking monoclonal antibody specific for complement component C5

AUTHOR(S): Wang, Yi; Hu, Qile; Madri, Joseph A.; Rollins, Scott A.; Chodera, Amy; Matis, Louis A.

LOCATION: Immumobiology Program, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA

JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1996 VOLUME: 93 NUMBER: 16 PAGES: 8563-8568 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English

14/3/2 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125084172 CA: 125(7)84172t JOURNAL
Expression of human CD59 in transgenic pig organs enhances organ survival in an ex vivo xenogeneic perfusion model
AUTHOR(S): Kroshus, Timothy J.; Bolman, R. Morton, III; Dalmasso, Agustin P.; Rollins, Scott A.; Guilmette, Edward R.; Williams, Barry L.; Squinto, Stephen P.; Fodor, William L.
LOCATION: Veterans Affairs Medical Center, University Minnesota, Minneapolis, MN, 55455, USA
JOURNAL: Transplantation DATE: 1996 VOLUME: 61 NUMBER: 10 PAGES: 1513-1521 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

14/3/3 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124325364 CA: 124(24)325364u PATENT
Retroviral transduction of cells using soluble complement inhibitors
INVENTOR(AUTHOR): Rother, Russell P.; Rollins, Scott A.; Mason, James M.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9603146 A1 DATE: 960208
APPLICATION: WO 95US8924 (950714) *US 278550 (940721)
PAGES: 49 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/395A
DESIGNATED COUNTRIES: AU; CA; JP DESIGNATED REGIONAL: AT; BE; CH; DE; DK ; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

14/3/4 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124315045 CA: 124(23)315045b PATENT
Methods for the preparation of retroviral particles and cell lines deficient in the .alpha.-galactosyl epitope
INVENTOR(AUTHOR): Rother, Russell P.; Rollins, Scott A.; Fodor, William L.; Springhorn, Jeremy P.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9603520 A1 DATE: 960208
APPLICATION: WO 95US8920 (950714) *US 278639 (940721) *US 399416 (950306)
PAGES: 101 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12P-021/06A; C12P-015/00B; C12P-007/04B; A61K-039/21B; C07H-021/04B
DESIGNATED COUNTRIES: AU; CA; JP; US DESIGNATED REGIONAL: AT; BE; CH; DE ; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

14/3/5 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124164049 CA: 124(13)164049c JOURNAL
Injectable retroviral particles for human gene therapy
AUTHOR(S): Squinto, Stephen P.; Rollins, Scott A.; Springhorn, Jeremy P.; Fodor, William L.; Rother, Russell P.
LOCATION: Alexion Pharmaceuticals, Inc., Haven, CT, 06511, USA
JOURNAL: Adv. Drug Delivery Rev. DATE: 1995 VOLUME: 17 NUMBER: 3
PAGES: 213-26 CODEN: ADDREP ISSN: 0169-409X LANGUAGE: English

14/3/6 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124143157 CA: 124(11)143157w JOURNAL
Monoclonal antibodies directed against human C5 and C8 block complement-mediated damage of xenogeneic cells and organs
AUTHOR(S): Rollins, Scott A.; Matis, Louis A.; Springhorn, Jeremy P.; Setter, Eva; Wolff, Dennis W.
LOCATION: Department of Immunobiology, Alexion Pharmaceuticals, Inc., New haven, CT, 06511, USA
JOURNAL: Transplantation DATE: 1995 VOLUME: 60 NUMBER: 11 PAGES: 1284-92 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

14/3/7 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124143156 CA: 124(11)143156v JOURNAL
Complement inhibition with an anti-C5 monoclonal antibody prevents acute cardiac tissue injury in an ex vivo model of pig-to-human xenotransplantation
AUTHOR(S): Kroshus, Timothy J.; Rollins, Scott A.; Dalmasso, Agustin P.; Elliott, Eileen A.; Matis, Louis A.; Squinto, Stephen P.; Bolman, R. Morton, III
LOCATION: Department of Surgery, University of Minnesota, Minneapolis, MN , USA
JOURNAL: Transplantation DATE: 1995 VOLUME: 60 NUMBER: 11 PAGES: 1194-202 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

14/3/8 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124127101 CA: 124(10)127101t PATENT
Anti-complement C5 antibodies for the treatment of glomerulonephritis and other inflammatory diseases
INVENTOR(AUTHOR): Evans, Mark J.; Matis, Louis; Mueller, Eileen Elliott; Nye, Steven H.; Rollins, Scott; Rother, Russell P.; Springhorn, Jeremy P.; Squinto, Stephen P.; Thomas, Thomas C.; et al.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9529697 A1 DATE: 951109
APPLICATION: WO 95US5688 (950501) *US 236208 (940502)
PAGES: 159 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-038/36A; A61K-039/00B; A61K-039/395B; C07K-014/00B; C07K-014/75B; C07K-016/00B; C07K-016/18B; C07K-016/36B; C07K-016/46B; C12N-005/10B; C12N-005/20B;

C12N-015/09B; C12N-015/10B; C12N-015/13B; C12N-015/63B; C12P-021/02B; C12P-021/08B DESIGNATED COUNTRIES: AM; AU; BB; BG; BR; BY; CA; CN; CZ; EE; FI; GE; HU; IS; JP; KG; KP; KR; KZ; LK; LR; LT; LV; MD; MG; MN; MX; NO; NZ; PL; RO; RU; SG; SI; SK; TJ; TM; TT; UA; UG; US; UZ; VN
DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG

14/3/9 (Item 9 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124114995 CA: 124(9)114995n JOURNAL
In vitro and in vivo inhibition of complement activity by a single-chain Fv fragment recognizing human C5
AUTHOR(S): Evans, Mark J.; Rollins, Scott A.; Wolff, Dennis W.; Rother, Russell P.; Norin, Allen J.; Therrien, Denise M.; Grijalva, Galo A.; Mueller, John P.; Nye, Steven H.; et al.
LOCATION: Dep. of Mol. Development, Alexion Pharmaceuticals, New Haven, CT, 06511, USA
JOURNAL: Mol. Immunol. DATE: 1995 VOLUME: 32 NUMBER: 16 PAGES: 1183-95 CODEN: MOIMD5 ISSN: 0161-5890 LANGUAGE: English

14/3/10 (Item 10 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124028050 CA: 124(3)28050u PATENT
Chimeric complement inhibitor proteins
INVENTOR(AUTHOR): Fodor, William L.; Rollins, Scott; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9523856 A1 DATE: 950908
APPLICATION: WO 95US2945 (950301) *US 205508 (940303)
PAGES: 86 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/00A; C07K-014/00B; C07H-021/00B DESIGNATED COUNTRIES: AU; BR; CA; CN; HU; JP; KR; MX; NO; NZ; RU DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR ; IE; IT; LU; MC; NL; PT; SE

14/3/11 (Item 11 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

124006626 CA: 124(1)6626j JOURNAL
Enzymic remodelling of the carbohydrate surface of a xenogenic cell substantially reduces human antibody binding and complement-mediated cytolysis
AUTHOR(S): Sandrin, Mauro S.; Fodor, William L.; Mouhtouris, Effie; Osman, Narin; Cohney, Shlomo; Rollins, Scott A.; Guilmette, Edward R.; Setter, Eva; Squinto, Stephen P.; et al.
LOCATION: Molecular Immunogenetics Lab., Austin Research Inst., Heidelberg, 3084, Australia
JOURNAL: Nat. Med. (N. Y.) DATE: 1995 VOLUME: 1 NUMBER: 12 PAGES: 1261-7 CODEN: NAMEFI ISSN: 1078-8956 LANGUAGE: English

14/3/12 (Item 12 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123337462 CA: 123(25)337462s PATENT
Method for reducing immune and hemostatic dysfunctions during extracorporeal circulation
INVENTOR(AUTHOR): Rollins, Scott A.; Smith, Brian R.; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.; Yale University
PATENT: PCT International ; WO 9525540 A1 DATE: 950928
APPLICATION: WO 95US3614 (950322) *US 217391 (940323)
PAGES: 34 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-039/00A; A61K-039/395B; C07K-016/00B; C07K-016/18B DESIGNATED COUNTRIES: AU; CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

14/3/13 (Item 13 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123312243 CA: 123(23)312243h PATENT
Recombinant preparation of terminal complement inhibitor fusion proteins lacking glycosyl-phosphatidylinositol (GPI) anchor and their use in organ transplantation
INVENTOR(AUTHOR): Rother, Russell P.; Rollins, Scott; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9523512 A1 DATE: 950908
APPLICATION: WO 95US2944 (950301) *US 205720 (940303)
PAGES: 85 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A01N-063/00A; A61K-035/14B; A61K-038/00B; C07H-017/00B; C07K-014/00B; C12N-001/00B; C12N-005/00B; C12N-005/06B; C12N-005/22B; C12N-007/01B; C12N-015/00B; C12N-015/03B; C12N-015/09B; C12N-015/06B; C12N-015/11B; C12P-100/00B; C12P-210/06B DESIGNATED COUNTRIES: AU; CA; JP DESIGNATED REGIONAL: AT; BE ; CH; DE; DK; ES; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE

14/3/14 (Item 14 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123283252 CA: 123(21)283252c JOURNAL
A novel bifunctional chimeric complement inhibitor that regulates C3 convertase and formation of the membrane attack complex
AUTHOR(S): Fodor, William L.; Rollins, Scott A.; Guilmette, Edward R.; Setter, Eva; Squinto, Stephen P.
LOCATION: Dep. Mol. Dev., Alexion Pharm., Inc., New Haven, CT, 06511, USA
JOURNAL: J. Immunol. DATE: 1995 VOLUME: 155 NUMBER: 9 PAGES: 4135-8
CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English

14/3/15 (Item 15 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123283240 CA: 123(21)283240x JOURNAL
A novel mechanism of retrovirus inactivation in human serum mediated by anti-.alpha.-galactosyl natural antibody
AUTHOR(S): Rother, Russell P.; Fodor, William L.; Springhorn, Jeremy P.; Birks, Carl W.; Setter, Eva; Sandrin, Mauro S.; Squinto, Stephen P.; Rollins, Scott A.
LOCATION: Departments Molecular Development Immunobiol., Alexion Pharmaceuticals, New Haven, CT, 06511, USA
JOURNAL: J. Exp. Med. DATE: 1995 VOLUME: 182 NUMBER: 5 PAGES: 1345-55
CODEN: JEMEAV ISSN: 0022-1007 LANGUAGE: English

14/3/16 (Item 16 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123245826 CA: 123(19)245826k JOURNAL
Complement-specific antibodies: designing novel anti-inflammatories
AUTHOR(S): Matis, Louis A.; Rollins, Scott A.
LOCATION: Immunobiol. Prog., Alexion Pharm., Inc., New Haven, CT, 06511, USA
JOURNAL: Nat. Med. (N. Y.) DATE: 1995 VOLUME: 1 NUMBER: 8 PAGES: 839-42 CODEN: NAMEFI ISSN: 1078-8956 LANGUAGE: English

14/3/17 (Item 17 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123225490 CA: 123(17)225490t JOURNAL
Blockade of C5a and C5b-9 generation inhibits leukocyte and platelet activation during extracorporeal circulation
AUTHOR(S): Rinder, Christine S.; Rinder, Henry M.; Smith, Brian R.; Fitch, Jane C. K.; Smith, Michael J.; Tracey, Jayne B.; Matis, Louis A.; Squinto, Stephen P.; Rollins, Scott A.
LOCATION: Dep. of Laboratory Medicine and Anesthesiology, Yale Univ. Sch. of Medicine and Yale-New Haven Hospital, New Haven, CT, 06510, USA
JOURNAL: J. Clin. Invest. DATE: 1995 VOLUME: 96 NUMBER: 3 PAGES: 1564-72 CODEN: JCINAO ISSN: 0021-9738 LANGUAGE: English

14/3/18 (Item 18 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123196481 CA: 123(15)196481h JOURNAL
Anti-C5 monoclonal antibody therapy prevents collagen-induced arthritis and ameliorates established disease
AUTHOR(S): Wang, Yi; Rollins, Scott A.; Madri, Joseph A.; Matis, Louis A.
LOCATION: Immunobiol. Program, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1995 VOLUME: 92 NUMBER: 19 PAGES: 8955-9 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English

14/3/19 (Item 19 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

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123141260 CA: 123(11)141260e JOURNAL
Rapid expression of an anti-human C5 chimeric Fab utilizing a vector that replicates in COS and 293 cells
AUTHOR(S): Evans, Mark J.; Hartman, Sandra L.; Wolff, Dennis W.; Rollins, Scott A.; Squinto, Stephen P.
LOCATION: Department of Molecular Development, Alexion Pharmaceuticals, Inc., 25 Science Park, New Haven, USA
JOURNAL: J. Immunol. Methods DATE: 1995 VOLUME: 184 NUMBER: 1 PAGES: 123-38 CODEN: JIMMBG ISSN: 0022-1759 LANGUAGE: English

14/3/20 (Item 20 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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123081609 CA: 123(7)81609p PATENT
Complement inhibitor proteins of non-human primates
INVENTOR(AUTHOR): Fodor, William L.; Rollins, Scott A.; Rother, Russel P. ; Squinto, Stephen P.
LOCATION: USA
ASSIGNEE: Alexion Pharmaceuticals, Inc.
PATENT: PCT International ; WO 9504756 A1 DATE: 950216
APPLICATION: WO 94US9046 (940810) *US 105735 (930811)
PAGES: 125 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C07K-014/435A; C07K-014/705B; A61K-038/17B; C12N-015/12B; C12N-015/79B
DESIGNATED COUNTRIES: JP DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR ; GB; GR; IE; IT; LU; MC; NL; PT; SE

14/3/21 (Item 21 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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123007584 CA: 123(1)7584k JOURNAL
The complement control protein homolog of herpesvirus saimiri regulates serum complement by inhibiting C3 convertase activity
AUTHOR(S): Fodor, William L.; Rollins, Scott A.; Bianco-Caron, Stella; Rother, Russell P.; Guilmette, Edward R.; Burton, Willis V.; Albrecht, Jens-Christian; Fleckenstein, Bernhard; Squinto, Stephen P.
LOCATION: Alexion Pharmaceuticals Inc., New Haven, CT, 06511, USA
JOURNAL: J. Virol. DATE: 1995 VOLUME: 69 NUMBER: 6 PAGES: 3889-92
CODEN: JOVIAM ISSN: 0022-538X LANGUAGE: English

14/3/22 (Item 22 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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122263065 CA: 122(21)263065v JOURNAL
Primate terminal complement inhibitor homologs of human CD59
AUTHOR(S): Fodor, William L.; Rollins, Scott A.; Bianco-Caron, Stella; Burton, Willis V.; Guilmette, Edward R.; Rother, Russell P.; Zavoico, George B.; Squinto, Stephen P.
LOCATION: Alexion Pharmaceuticals, Inc., New Haven, CT, 06511-1968, USA
JOURNAL: Immunogenetics DATE: 1995 VOLUME: 41 NUMBER: 1 PAGES: 51
CODEN: IMNGBK ISSN: 0093-7711 LANGUAGE: English

14/3/23 (Item 23 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122158376 CA: 122(13)158376z JOURNAL
Expression of recombinant transmembrane CD59 in paroxysmal nocturnal hemoglobinuria B cells confers resistance to human complement
AUTHOR(S): Rother, Russell P.; Rollins, Scott A.; Mennone, John; Chodera, Amy; Fidel, Seth A.; Bessler, Monica; Hillmen, Peter; Squinto, Stephen P.
LOCATION: Alexion Pharmaceuticals Inc., New Haven, CT, USA
JOURNAL: Blood DATE: 1994 VOLUME: 84 NUMBER: 8 PAGES: 2604-11
CODEN: BLOOAW ISSN: 0006-4971 LANGUAGE: English

14/3/24 (Item 24 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122053923 CA: 122(5)53923x JOURNAL
Molecular and functional analysis of porcine E-selectin reveals a potential role in xenograft rejection
AUTHOR(S): Rollins, Scott A.; Evans, Mark J.; Johnson, Krista K.; Elliot, Eileen A.; Squinto, Steven P.; Matis, Louis A.; Rother, Russell P.
LOCATION: Dep. Immunobiol., Alexion Pharmaceuticals Inc., New Haven, CT, 06511, USA
JOURNAL: Biochem. Biophys. Res. Commun. DATE: 1994 VOLUME: 204
NUMBER: 2 PAGES: 763-71 CODEN: BBRCA9 ISSN: 0006-291X LANGUAGE: English

14/3/25 (Item 25 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121298801 CA: 121(25)298801p JOURNAL
Expression of a functional human complement inhibitor in a transgenic pig as a model for the prevention of xenogeneic hyperacute organ rejection
AUTHOR(S): Fodor, William L.; Williams, Barry L.; Matis, Louis A.; Madri, Joseph A.; Rollins, Scott A.; Knight, James W.; Velander, William; Squinto, Stephen P.
LOCATION: Alexion Pharm. Inc., New Haven, CT, 06511, USA
JOURNAL: Proc. Natl. Acad. Sci. U. S. A. DATE: 1994 VOLUME: 91
NUMBER: 23 PAGES: 11153-7 CODEN: PNASA6 ISSN: 0027-8424 LANGUAGE: English

14/3/26 (Item 26 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121203257 CA: 121(17)203257d JOURNAL
Evidence that activation of human T cells by porcine endothelium involves direct recognition of porcine SLA and costimulation by porcine ligands for LFA-1 and CD2
AUTHOR(S): Rollins, Scott A.; Kennedy, Scott P.; Chodera, Amy J.; Elliott, Eileen A.; Zavoico, George B.; Matis, Louis A.

LOCATION: Department of Immunobiology, Alexion Pharmaceuticals, Inc., New Haven, CT, 06511, USA
JOURNAL: Transplantation DATE: 1994 VOLUME: 57 NUMBER: 12 PAGES: 1709-16 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

14/3/27 (Item 27 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

121131942 CA: 121(11)131942y JOURNAL
Protection of porcine aortic endothelial cells from complement-mediated cell lysis and activation by recombinant human CD59
AUTHOR(S): Kennedy, Scott P.; Rollins, Scott A.; Burton, Willis V.; Sims, Peter J.; Bothwell, Alfred L. M.; Squinto, Stephen P.; Zavoico, George B.
LOCATION: Dep. Vasc. Biol., Alexion Pharm. Inc., New Haven, CT, 06511, USA
JOURNAL: Transplantation DATE: 1994 VOLUME: 57 NUMBER: 10 PAGES: 1494-501 CODEN: TRPLAU ISSN: 0041-1337 LANGUAGE: English

14/3/28 (Item 28 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120101475 CA: 120(9)101475k JOURNAL
Inhibition of complement-mediated cytolysis by the terminal complement inhibitor of herpesvirus saimiri
AUTHOR(S): Rother, Russell P.; Rollins, Scott A.; Fodor, William L.; Albrecht, Jens C.; Setter, Eva; Fleckenstein, Bernhard; Squinto, Stephen P.
LOCATION: Alexion Pharm. Inc., New Haven, CT, 06511, USA
JOURNAL: J. Virol. DATE: 1994 VOLUME: 68 NUMBER: 2 PAGES: 730-7
CODEN: JOVIAM ISSN: 0022-538X LANGUAGE: English

14/3/29 (Item 29 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

118198171 CA: 118(20)198171c PATENT
Genetically engineered cells as universal donor cells for vascular grafts or drug delivery
INVENTOR(AUTHOR): Sims, Peter J.; Bothwell, Alfred L. M.; Elliot, Eileen A.; Flavell, Richard A.; Madri, Joseph; Rollins, Scott; Bell, Leonard; Squinto, Stephen
LOCATION: USA
ASSIGNEE: Oklahoma Medical Research Foundation; Yale University
PATENT: PCT International ; WO 9302188 A1 DATE: 930204
APPLICATION: WO 92US5920 (920714) *US 729926 (910715) *US 906394 (920629)
PAGES: 88 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/00A; C12N-015/12B; A01K-067/027B; C12N-005/16B; C12N-005/22B; C12N-015/87B; A61L-027/00B; C07K-015/00B DESIGNATED COUNTRIES: CA; JP
DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LU; MC; NL; SE

14/3/30 (Item 30 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

116253695 CA: 116(25)253695n JOURNAL
Contribution of the N-linked carbohydrate of erythrocyte antigen CD59 to its complement-inhibitory activity
AUTHOR(S): Ninomiya, Haruhiko; Stewart, Betty H.; Rollins, Scott A.; Zhao, Ji; Bothwell, Alfred L. M.; Sims, Peter J.
LOCATION: Health Sci. Cent., Univ. Oklahoma, Oklahoma City, OK, 73104, USA
JOURNAL: J. Biol. Chem. DATE: 1992 VOLUME: 267 NUMBER: 12 PAGES: 8404-10 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English

14/3/31 (Item 31 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

115277572 CA: 115(25)277572a DISSERTATION
Isolation and characterization of CD59, a membrane attack complex inhibitor of complement
AUTHOR(S): Rollins, Scott Alan
LOCATION: Univ. Oklahoma Health Sci. Cent., Norman, OK, USA
DATE: 1990 PAGES: 192 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int. B 1991, 51(12, Pt. 1), 5802 AVAIL: Univ. Microfilms Int., Order No. DA9113763

14/3/32 (Item 32 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

115133684 CA: 115(13)133684r JOURNAL
Inhibition of homologous complement by CD59 is mediated by a species-selective recognition conferred through binding to C8 within C5b-8 or C9 within C5b-9
AUTHOR(S): Rollins, Scott A.; Zhao, Ji; Ninomiya, Haruhiko; Sims, Peter J.
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
JOURNAL: J. Immunol. DATE: 1991 VOLUME: 146 NUMBER: 7 PAGES: 2345-51
CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English

14/3/33 (Item 33 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

115129040 CA: 115(13)129040k JOURNAL
Amplified gene expression in CD59-transfected Chinese hamster ovary cells confers protection against the membrane attack complex of human complement
AUTHOR(S): Zhao, Ji; Rollins, Scott A.; Maher, Stephen E.; Bothwell, Alfred L. M.; Sims, Peter J.
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
JOURNAL: J. Biol. Chem. DATE: 1991 VOLUME: 266 NUMBER: 20 PAGES: 13418-22 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English

14/3/34 (Item 34 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

114180121 CA: 114(19)180121u JOURNAL
DNA ploidy and p21 protein levels in tissue sections as end-point markers in animal carcinogenesis trials
AUTHOR(S): Rhodes, Steven W.; Hurst, Robert E.; Rollins, Scott A.; Jones, Philip L.; Hemstreet, George P.; Detrisac, Carol J.; Thomas, Cathy F.; Moon, Richard C.; Kelloff, Gary J.
LOCATION: Health Sci. Cent., Univ. Oklahoma, Oklahoma City, OK, 73190, USA
JOURNAL: Biol. Monit. DATE: 1991 VOLUME: 1 NUMBER: 1 PAGES: 61-73
CODEN: BIMNE2 LANGUAGE: English

14/3/35 (Item 35 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

114099628 CA: 114(11)99628t JOURNAL
Regulatory control of the terminal complement proteins at the surface of human endothelial cells: neutralization of a C5b-9 inhibitor by antibody to CD59
AUTHOR(S): Hamilton, Karen K.; Ji, Zhao; Rollins, Scott; Stewart, Betty H.; Sims, Peter J.
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, USA
JOURNAL: Blood DATE: 1990 VOLUME: 76 NUMBER: 12 PAGES: 2572-7
CODEN: BLOOAW ISSN: 0006-4971 LANGUAGE: English

14/3/36 (Item 36 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

113057087 CA: 113(7)57087q JOURNAL
The complement-inhibitory activity of CD59 resides in its capacity to block incorporation of C9 into membrane C5b-9
AUTHOR(S): Rollins, Scott A.; Sims, Peter J.
LOCATION: Health Sci. Cent., Oklahoma Univ., Oklahoma City, OK, 73104, USA
JOURNAL: J. Immunol. DATE: 1990 VOLUME: 144 NUMBER: 9 PAGES: 3478-83
CODEN: JOIMA3 ISSN: 0022-1767 LANGUAGE: English

14/3/37 (Item 37 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

111230335 CA: 111(25)230335c JOURNAL
Regulatory control of complement on blood platelets. Modulation of platelet procoagulant responses by a membrane inhibitor of the C5b-9 complex
AUTHOR(S): Sims, Peter J.; Rollins, Scott A.; Wiedmer, Therese
LOCATION: Cardiovasc. Biol. Res. Program, Oklahoma Med. Res. Found., Oklahoma City, OK, 73104, USA
JOURNAL: J. Biol. Chem. DATE: 1989 VOLUME: 264 NUMBER: 32 PAGES:

19228-35 CODEN: JBCHA3 ISSN: 0021-9258 LANGUAGE: English
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